REVIEW OF APPROACHES FOR SETTING AN OBJECTIVE FOR MIXTURES IN AMBIENT AIR USING POLYCYCLIC AROMATIC HYDROCARBONS (PAHs)

As an example:

BENZO[a]PYRENE

Alberta Environment

Review of Approaches for Setting an Objective for Mixtures in Ambient Air using Polycyclic Aromatic Hydrocarbons (PAHs) as an Example:

Benzo[a]pyrene

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FOREWORD

Alberta Environment maintains Ambient Air Quality Objectives to support air quality management in Alberta. Currently Alberta Environment has ambient objectives for more than forty substances. These objectives are periodically updated and new objectives are developed as required.

With the assistance of the Clean Air Strategic Alliance, a multi-stakeholder workshop was held in October 2004 to *set Al*berta's priorities for the next three years. Based on those recommendations to Alberta Environment, a three-year work plan was developed to review four existing objectives, and create three new objectives.

This document is one in a series of documents that presents the scientific assessment for these substances.

Long Fu
Project Coordinator
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TABLE OF CONTENTS

LIST OF TABLES ..... **v**
LIST OF FIGURES ..... **vi**
SUMMARY ..... **vii**

1.0 INTRODUCTION **1**

2.0 GENERAL SUBSTANCE INFORMATION **2**
2.1 Physical and Chemical Properties 2
2.2 Emission Sources and Ambient Levels 4
2.2.1 Natural Sources *4*
2.2.2 Anthropogenic Sources *4*
2.2.3 Ambient Levels *4*

3.0 ATMOSPHERIC CHEMISTRY AND FATE **8**

4.0 EFFECTS ON HUMANS **9**
4.1 Overview of Chemical Disposition 9
4.2 Genotoxicity and Carcinogenicity 10
4.3 Acute and Sub-Acute Effects 12
4.3.1 Acute Human Effects *12*
4.3.2 Acute and Sub-Acute Animal Effects *12*
4.3.2.1 Ingestion **12**
4.3.2.1.1 Reproductive and Developmental Toxicity 14
4.3.2.1.2 Genotoxicity and Carcinogenicity 15
4.3.2.2 Other Routes and Effects **15**
4.4 Chronic Effects 16
4.4.1 Chronic Human Effects *16*
4.4.1.1 Inhalation **16**
4.4.1.1.1 Respiratory 16
4.4.1.1.2 Immunological and Lymphoreticular Effects 18
4.4.1.1.3 Carcinogenicity 18
4.4.1.2 Ingestion **18**
4.4.1.3 Other Routes and Effects **18**
4.4.2 Sub-Chronic and Chronic Animal Effects *18*
4.4.2.1.1 Inhalation 19
4.4.2.1.2 Death 19
4.4.2.1.3 Carcinogenicity 19
4.4.2.2 Ingestion **20**
4.4.2.2.1 Carcinogenicity 20
4.4.2.3 Other Routes and Effects **21**
4.5 Other Effects 21
4.6 Summary Of Adverse Health Effects Of Benzo[a]Pyrene 21

5.0 EFFECTS ON VEGETATION AN MATERIALS **23**

5.1 Vegetation 23
5.2 Materials 24

6.0 AIR SAMPLING AND ANALYTICAL METHODS 26
6.1 Reference Methods 26
6.1.1 US EPA Compendium Method TO-13A 26
6.1.2 NIOSH Method 5506 26
6.1.3 NIOSH Method 5515 27
6.2 Alternative, Emerging Techniques 27
6.2.1 Alternative Techniques 28
6.2.1.1 Sampling 28
6.2.1.2 Extraction 29
6.2.1.3 Cleanup 30
6.2.1.4 Analysis 30
6.2.2 Emerging Techniques 31

7.0 AMBIENT OBJECTIVES AND GUIDELINES 34
7.1 Benzo[a]pyrene Air Quality Guidelines 34
7.1.1 Canada 34
7.1.2 United States 34
7.1.3 International Agencies 35

8.0 RISK CHARACTERIZATION 37
8.1 Relevant Chemical Forms 37
8.2 Exposure Assessment 37
8.3 Toxicity Assessment 38
8.3.1 Mixtures of Carcinogenic PAHs 39
8.4 Characterization of Risk 41

9.0 REFERENCES 43

APPENDIX A 53
APPENDIX B 61

LIST OF TABLES

Table 1 Identification of Benzo[a]pyrene[a] 2

Table 2 Physical and Chemical Properties for Benzo[a]pyrene[a] 3

Table 3 Alberta Emissions of Benzo[a]pyrene According to the 2001 NPRI Database (NPRI, 2004) 6

Table 4 Alberta Air Emissions of Benzo[a]pyrene According to the 2001 NPRI Database (NPRI, 2004) 6

Table 5 Annual Average Concentrations of Selected PAHs in AmbientAir at Various Sites in Alberta (1994 to 2000) 7

Table 6 Examples of NOAELs and LOAELs Associated with Acute and Sub-Acute Benzo[a]pyrene Ingestion (Experimental Animals) 13

Table 7 Examples of NOAELs and LOAELs Associated with Sub-Chronic and Chronic Benzo[a]pyrene Inhalation (Human). 17

Table 8 Examples of NOAELs and LOAELs Associated with Sub-Chronic and Chronic Benzo[a]pyrene Inhalation (Experimental Animals) 19

Table 9 Examples of NOAELs and LOAELs Associated with Sub-Chronic Benzo[a]pyrene Ingestion (Experimental Animals) 20

Table 10 Method Advantages and Disadvantages 28

Table 11 Summary of Air Quality Guidelines for Benzo[a]pyrene 36

Table 12 Relative Potency of Individual PAHs to Benzo[a]pyrene (Toxicity Equivalent Factor – TEF – Method) 39

LIST OF FIGURES

Figure 1 Range of Air Quality Guidelines for Benzo[a]pyrene Proposed by Various Agencies for Protection of Human Receptors 42

SUMMARY

Benzo[a]pyrene is the most well known polycyclic aromatic hydrocarbon (PAH) in a large group of organic compounds with two or more fused aromatic rings. PAHs are formed mainly as a result of incomplete combustion of organic materials during industrial and other human activities. These activities include processing of coal and crude oil, combustion of natural gas, combustion of refuse, vehicle traffic, cooking and tobacco smoking, as well as natural processes such as forest fires. Motor vehicle exhaust and re-suspension are major contributors of PAHs, including benzo[a]pyrene, to urban air. PAHs in urban air have also been associated with residential wood burning emissions.

Benzo[a]pyrene will tend to be incorporated onto particulates during cooling and condensation in the atmosphere. Consequently, benzo[a]pyrene will generally exist in the particle phase at normal ambient temperatures in the atmosphere. Particle sizes will be mostly ≤ 2.5 µm in aerodynamic diameter. Processes governing the fate of benzo[a]pyrene in the atmosphere are the same processes that govern transport and removal of these small particles from the atmosphere.

Primary endpoints of toxicity associated with chronic benzo[a]pyrene exposures at doses >1 mg/m^3 in animals are carcinogenicity, reproductive toxicity, and developmental toxicity. Other non-carcinogenic effects reported at doses >100 mg/m^3 to animals include growth inhibition, immunosuppression, liver damage, and irritation/hypersensitivities. Severity of the effects depends on dose, administration of dose (route and vehicle of administration), and animal species, age and genotype. Epidemiological studies in coke-oven, coal-gas, and aluminum production workers have provided evidence of the role of inhaled benzo[a]pyrene at doses >0.1 mg/m^3 in the causation of lung cancers.

PAHs are common contaminants of terrestrial and aquatic ecosystems. Plants acquire PAHs, including benzo[a]pyrene, by absorption through their roots. Once entry has occurred, it is thought that PAHs can then be translocated to all plant tissues. PAHs can also enter the plant by foliar deposition. Toxicological studies for defining potential hazards of PAHs have been conducted in the absence of UV radiation. Current evidence indicates that coexposure of UV light and near UV light and specific PAHs, including benzo[a]pyrene, can result in increased toxicity to plants.

Inhalation exposure to single PAH compounds, for example benzo[a]pyrene alone, does not occur without other PAHs being present. Several PAHs with four of more rings are treated as having the potential to cause cancer in addition to benzo[a]pyrene. As a result, benzo[a]pyrene is proposed as an indicator for the carcinogenic fraction of these PAHs which are all present as mixtures in ambient air. Further, a method using factors of 10 to represent the potency of individual PAHs relative to benzo[a]pyrene is recommended to address mixtures of PAHs in ambient air.

The ability of benzo[a]pyrene and other PAHs to contribute to health effects in the general human population as a result of low-level environmental exposure is much less certain than what has been reported in animal studies and in the workplace. As a result regulatory agencies adopt

air quality guidelines for benzo[a]pyrene and other PAHs in order to protect humans. Benzo[a]pyrene concentrations in indoor air, in air outside of homes, and in urban (city) air reported in scientific literature tend to be within the range of concentrations representing air quality guidelines developed by regulatory agencies for protection of human receptors.

1.0 INTRODUCTION

Alberta Environment establishes Ambient Air Quality Guidelines under Section 14 of the Environmental Protection and Enhancement Act (EPEA). These guidelines are part of the Alberta air quality management system (AENV, 2000).

The main objective of this report was to provide a review of scientific and technical information to assist in evaluating the basis and background for an ambient air quality guideline for benzo[a]pyrene. The following aspects were examined as part of the review:

- physical and chemical properties
- existing and potential anthropogenic emissions sources in Alberta
- effects on humans, animals, vegetation, and materials
- ambient air guidelines in other Canadian jurisdictions, United States, World Health Organization and New Zealand, and the basis for development and use
- characterization of risks to exposed receptors
- monitoring techniques

Important physical and chemical properties that govern the behaviour of benzo[a]pyrene in the environment were reviewed and presented in this report. Existing and potential anthropogenic sources of benzo[a]pyrene emissions in Alberta were also presented. Anthropogenic emissions are provided in Environment Canada's National Pollutant Release Inventory (NPRI).

Scientific information about the effects of benzo[a]pyrene on humans and animals is reported in published literature and other sources. This information includes toxicological studies published in professional journals and reviews and information available through the US Agency for Toxic Substances and Disease Registry (ATSDR) and US Environmental Protection Agency's Integrated Risk Information System (IRIS). These sources provided valuable information for understanding health effects of benzo[a]pyrene exposure.

Ambient air guidelines for benzo[a]pyrene are used by numerous jurisdictions in North America for different averaging-time periods. These guidelines are developed using cancer risk assessment procedures. The basis for how these approaches are used by different jurisdiction to develop guidelines was investigated in this report.

Accurate measurement of polycyclic aromatic compounds, including benzo[a]pyrene, in ambient air can be challenging in part because of a variety of potential techniques for sampling and analysis, and the lack of standardized and documented methods. The United States Environmental Protection Agency (US EPA), National Institute of Occupational Safety and Health (NIOSH), and Occupational Safety and Health Administration (OSHA) are the only organizations that provide documented and technically reviewed methodologies for determining concentrations of benzo[a]pyrene in ambient and indoor air. These methods, which are generally accepted as the preferred methods, were reviewed and presented in this report.

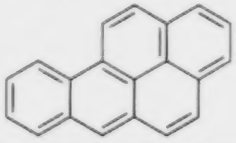
2.0 GENERAL SUBSTANCE INFORMATION

Benzo[a]pyrene is a five-ring polycyclic aromatic hydrocarbon (PAH) which exists as a pale yellow solid with an aromatic odour at ambient pressures and temperatures (Genium, 1999). Benzo[a]pyrene will burn but will not readily ignite (Genium, 1999). It is stable but incompatible with strong oxidizers and oxidizing chemicals (Genium, 1999). Decomposition products of benzo[a]pyrene include carbon monoxide and carbon dioxide (Genium, 1999). Table 1 provides a list of important identification numbers and common synonyms for benzo[a]pyrene.

2.1 Physical and Chemical Properties

The physical and chemical properties of benzo[a]pyrene are summarized in Table 2.

Table 1 Identification of Benzo[a]pyrene[a]

Property	
Chemical Formula	$C_{20}H_{12}$
Chemical Structure	
CAS Registry number	50-32-8
RTECS number	DJ3675000
UN Number	No data
Common Synonyms and Tradenames	B(a)P BAP Benzo(D,E,F)chrysene 3,4-benzopirene 3,4-benzopyrene 1,2-benzopyrene 6,7-benzopyrene 3,4-benzpyren 3,4-benz(a)pyrene 3,4-benzpyrene benz(a)pyrene 3,4-benzylpyrene 3,4-benzypyrene 3,4-bp Bp Coal tar pitch volatiles: Benzo(a)pyrene

[a] all data from Genium (1999) unless otherwise stated

Table 2 Physical and Chemical Properties for Benzo[a]pyrene[a]

Property	Value
Molecular weight (g/mol)	252.3
Physical state	pale yellow solid (plates and needles)
Melting point (°C)	181.1
Boiling point (°C)	495[b]
Density (g/cm^3)	1.351[c]
Vapour pressure	> 1mm of Hg at 20°C 0.37×10^{-6} mPa at 20°C[f]
Vapour density	8.7
Solubility in water (% mass)	3×10^{-7} (at T=25°C) (insoluble)
Solubility in other solvents	very soluble in chloroform; soluble in benzene, toluene and xylene[b]
Henry's Law Constant ($kPa.m^3/mol$)	4.65×10^{-5} (at T=25°C)
Octanol water partition coefficient (log K_{ow})	6.20
Organic carbon partition coefficient (log K_{oc})	4.0 to 8.3[e]
Bioconcentration factor (log BCF)	-0.155 to 6.95[e]
Odour	faint aromatic odour[c,d]
Odour threshold (mg/m^3)	no data
Flash point	non-flammable[c]
Conversion factors for vapour[g] (at 25 °C and 101.3 kPa)	1 ppb = 10.3 $\mu g/m^3$ 1 $\mu g/m^3$ = 0.0969 ppb

[a] all data from Lide, 2004 unless otherwise indicated
[b] RSC, 1999
[c] Genium
[d] ATSDR, 1995
[e] Mackay *et al.*, 1992
[f] EC, 1994
[g] WHO, 1998

2.2 Emission Sources and Ambient Levels

2.2.1 Natural Sources

Natural sources of PAHs and hence of benzo[a]pyrene include forest fires, volcanic eruptions, diagenisis and biosythesis (EC, 1994). According to a 1990 inventory of PAH emissions in Canada, forest fires accounted for 46% of the total PAHs emissions to the atmosphere (LGL, 1993 as cited in EC, 1994). It is believed however that the emissions from forest fires have a small impact on ambient PAH levels. PAHs also occur naturally in coal derivatives and petroleum (NRCC, 1983 as cited in EC, 1994). Benzo[a]pyrene may be synthesized by various bacteria and algae (Verschueren, 2001).

2.2.2 Anthropogenic Sources

According to IARC (1983) (cited in HSDB, 2004), benzo[a]pyrene is not commercially produced or used. Benzo[a]pyrene occurs in the environment ubiquitously as the result of incomplete combustion of fuels and other organic materials such as motor oils, gasoline, tobacco, cooking oils, butter, margarine, and other food (IARC, cited in HSDB, 2004). Benzo[a]pyrene is therefore emitted in tobacco smoke and vehicle exhaust (gasoline and diesel engine exhaust) (IARC, cited in HSDB, 2004).

Anthropogenic activities leading to releases of PAHs to the atmosphere therefore include activities such as aluminum smelting, residential wood combustion, agricultural burning and open air fires and transportation (diesel combustion) (EC, 1998; 1994).

According to the National Pollutant Release Inventory (NPRI), the industrial sectors contributing to benzo[a]pyrene emissions in Alberta are the crude petroleum and natural gas industry, the refined petroleum and coal products sector, the paper and allied products industries and the chemical and chemical products sector (NPRI, 2004). Table 3 provides the total emissions of benzo[a]pyrene (in kg) for Alberta as reported in the 2001 NPRI database (NPRI, 2004). Table 4 presents the air emissions of benzo[a]pyrene (in kg) in Alberta as reported in the 2001 NPRI database (NPRI, 2004). Data for total emissions of benzo[a]pyrene for all provinces are presented in Appendix A. It should be noted that the emissions reported in Tables 2.3 and 2.4 and in Appendix A are for benzo[a]pyrene as an individual compound. In theNPRI database, benzo[a]pyrene is also included (with 16 other substances) as PAHs under Schedule 1, Part 3 (NPRI, 2001) however this emissions data was not used in this report.

2.2.3 Ambient Levels

Ambient levels of benzo[a]pyrene in the atmosphere are reviewed in Verschueren (2001), Eisler (2000), EC (1998, 1994) and ATSDR (1995). In addition, the Clean Air Strategic Alliance (CASA) also monitors benzo[a]pyrene as PAHs in ambient air.

A number of studies have investigated the concentrations of PAHs, including benzo[a]pyrene, in indoor and outdoor air and at different times of the year (summer versus winter). Studies, such as the recent studies by Ohura *et al.* (2004) and Zhu *et al.* (2003), and other studies cited in the above mentioned reviews, have shown that the outdoor air levels of PAHs are seasonal and that they tend to vary with meteorological conditions and variations in emissions from seasonal sources such as heating (Ohura *et al.*, 2004). Indoor air concentrations of PAHs, including benzo[a]pyrene, were affected by activities such as smoking, the use of kerosene space heaters and the use of PAH containing products such as insect repellants. Indoor air concentrations were also affected by the age of house, the type of house (wood) and the outdoor PAH concentrations (Ohura *et al.*, 2004).

Annual average concentrations of selected PAHs in ambient air were available from several monitoring sites in Alberta for the period 1994 to 2000 (Myrick, 2004). These concentrations are shown in Table 5. The monitoring sites included three Alberta Environment air-monitoring stations in Calgary, three in Edmonton, one in Fort Saskatchewan, and one former rural site 10 km northwest of Vegreville. PAHs measured during this period included benzo[a]pyrene, benzo[b,j,k]fluoranthene, benzo[e]pyrene, benzo[ghi]perylene, and indeno[123cd]pyrene.

Table 3 Alberta Emissions of Benzo[a]pyrene According to the 2001 NPRI Database (NPRI, 2004)

NPRI ID	Company	City	Province	Alberta Emissions of Benzo[a]pyrene (kg)				
				Air	Water	Land	Underground	Total
3903	Petro-Canada - Edmonton Refinery	Edmonton	AB	0.169	0	0	301.543	301.712
3707	Imperial Oil - Strathcona Refinery	Edmonton	AB	1.515	0	14.91	0	16.425
2875	Weyerhaeuser Company Ltd.	Grande Prairie	AB	0.749	0	2.584	0	3.333
2274	Syncrude Canada Ltd. - Mildred Lake Plant Site	Fort McMurray	AB	0.903	0	0	0	0.903
2960	Shell Canada Products - Shell Scotford Refinery	Fort Saskatchewan	AB	0.359	0	0	0	0.359
5357	Cancarb Limited - Cancarb	Medicine Hat	AB	0.200	0	0	0	0.200
				3.895	0	17.494	301.543	322.932

Table 4 Alberta Air Emissions of Benzo[a]pyrene According to the 2001 NPRI Database (NPRI, 2004)

NPRI ID	Company	City	Province	Alberta Air Emissions of Benzo[a]pyrene (kg)					
				Stack /Point	Storage /Handling	Fugitive	Spills	Other Non-Point	Total
3903	Petro-Canada - Edmonton Refinery	Edmonton	AB	0	0	0.169	0	0	0.169
3707	Imperial Oil - Strathcona Refinery	Edmonton	AB	0.471	0.157	0.868	0	0.019	1.515
2875	Weyerhaeuser Company Ltd.	Grande Prairie	AB	0.749	0	0	0	0	0.749
2274	Syncrude Canada Ltd. - Mildred Lake Plant Site	Fort McMurray	AB	0.903	0	0	0	0	0.903
2960	Shell Canada Products - Shell Scotford Refinery	Fort Saskatchewan	AB	0.149	0.073	0.137	0	0	0.359
5357	Cancarb Limited - Cancarb	Medicine Hat	AB	0.2	0	0	0	0	0.200
				2.472	0.230	1.174	0	0.019	3.895

Table 5 **Annual Average Concentrations of Selected PAHs in AmbientAir at Various Sites in Alberta (1994 to 2000)**

Year	B[a]P	BF	B[e]P	BP	IP
	Calgary Central				
1994	0.37	no data	no data	no data	no data
1995	0.17	no data	no data	no data	no data
1996	0.17	0.29	0.45	0.77	0.26
1997	0.12	0.20	0.18	0.46	0.22
1998	0.20	0.39	0.23	0.65	0.30
1999	0.10	0.26	0.15	0.39	0.20
2000	0.08	0.23	0.13	0.28	0.13
	Calgary East				
1994	0.24	no data	no data	no data	no data
1995	0.21	no data	no data	no data	no data
1996	0.21	0.44	0.47	0.75	0.32
1997	0.16	0.32	0.29	0.59	0.32
1998	0.11	0.26	0.14	0.26	0.16
1999	0.13	0.37	0.23	0.52	0.27
2000	0.12	0.34	0.20	0.40	0.20
	Calgary Northwest				
1994	0.20	no data	no data	no data	no data
1995	0.09	no data	no data	no data	no data
1996	0.07	0.17	0.21	0.28	0.13
1997	0.04	0.11	0.09	0.22	0.13
1998	0.07	0.23	0.11	0.27	0.17
1999	0.04	0.14	0.08	0.19	0.11
2000	0.05	0.16	0.08	0.15	0.09
	Edmonton Central				
1994	0.37	no data	no data	no data	no data
1995	0.20	no data	no data	no data	no data
1996	0.20	0.38	0.34	0.70	0.28
1997	0.14	0.24	0.21	0.50	0.25
1998	0.19	0.44	0.23	0.53	0.30
1999	0.14	0.32	0.17	0.39	0.22
2000	0.12	0.29	0.16	0.35	0.18
	Edmonton East				
1994	0.18	no data	no data	no data	no data
1995	0.12	no data	no data	no data	no data
1996	0.11	0.23	0.32	0.34	0.16
1997	0.09	0.18	0.14	0.25	0.16
1998	0.11	0.26	0.14	0.26	0.16
1999	0.09	0.22	0.12	0.23	0.15
2000	0.10	0.24	0.13	0.25	0.13
	Edmonton Northwest				
1994	0.41	no data	no data	no data	no data
1995	0.24	no data	no data	no data	no data
1996	0.20	0.37	0.53	0.71	0.31
1997	0.16	0.27	0.24	0.55	0.28
1998	0.22	0.50	0.27	0.63	0.33
1999	0.13	0.33	0.19	0.47	0.26
2000	0.14	0.35	0.19	0.40	0.20
	Fort Saskatchewan				
1994	0.16	no data	no data	no data	no data
1995	0.09	no data	no data	no data	no data
1996	0.07	0.15	0.21	0.23	0.12
1997	0.06	0.13	0.09	0.20	0.12
1998	0.08	0.22	0.11	0.23	0.14
1999	0.05	0.15	0.08	0.17	0.11
2000	0.08	0.22	0.10	0.19	0.13
	Royal Park (10 km northwest of Vegreville)				
1994	0.01	no data	no data	no data	no data
1995	0.01	no data	no data	no data	no data
1996	0.01	0.03	0.06	0.03	0.02
1997	0.00	0.02	0.01	0.02	0.02

Note: Concentration (ng/m^3), benzo[a]pyrene (B[a]P), benzo[b,j,k]fluoranthene (BF), benzo[e]pyrene (B[e]P), benzo[ghi]perylene (BP), indeno[123cd]pyrene (IP).
Source: Myrick (2004).

3.0 ATMOSPHERIC CHEMISTRY AND FATE

When benzo[a]pyrene is released to the atmosphere, it may undergo (i) reaction with species such as ozone (O_3), nitrous oxide (NO_2) and photochemically produced hydroxyl (OH) radicals, (ii) direct photolysis and (iii) adsorption to particulate matter. For reactions with O_3, NO_2 and OH radicals, the reported half-lives are 37 minutes, 7 days and 22 hours, respectively (Genium, 1999).

The vapour pressure of the PAH is an important chemical property when determining whether a PAH will absorb to particulate matter or remain in the vapour phase (EC, 1994). For PAHs with low vapour pressures and are emitted from combustion or other high temperature sources (as is the case with benzo[a]pyrene), these PAHs are typically associated with particulate matter (EC, 1994) of small size (<1μm) (Sheu *et al.*, 1997).

Once associated with the particulate matter, the fate of benzo[a]pyrene in the atmosphere will be related to the fate of the particulate matter it is associated with and will be influenced by the particle size, the meteorological conditions and atmospheric physics (Eisler, 2000). PAHs associated with particulate matter can be transported over large distances (Lunde and Bjørseth, Neff, NRCC, cited in EC, 1994) before being removed by wet deposition, dry depositions and chemical transformation (Neff, NRCC, Van Noort and Wondergem, Ligocki *et al.*, cited in EC, 1994). For wet and dry deposition, dry deposition accounts for most of the removal of benzo[a]pyrene (ATSDR, 1995). Chemical transformations, which include photolysis and reaction with oxidant gases (ATSDR, 1995), are influenced by the nature of the particulate matter to which the PAH is absorbed (i.e. organic content) (Korfmacher, NRCC, Behymer and Rites, cited in EC, 1994) and by the concentration of PAH absorbed (Kamens *et al.*, Slooff *et al.*, cited in EC, 1994).

4.0 EFFECTS ON HUMANS

Air-borne polyaromatic hydrocarbons (PAHs) have been measured in coking, coal tar, and asphalt production emissions; smokehouses; municipal trash incinerator emissions; cigarette and wood smoke; automobile exhaust; asphalt roads, or agriculture burns. Polyaromatic hydrocarbons (PAHs) are also a common contaminant at hazardous waste sites, can occur after processing or cooking specific foods; and, may occur as a contaminant in food, milk, or water (ATSDR, 1996; Klaasen, Amdur and Doull, 1996).

Benzo[a]pyrene (B[a]P) is one of the most studied PAHs. It is classified as an animal carcinogen and a probable human carcinogen (IARC, 1983; IRIS, 1994; ATSDR, 1995; ACGIH, cited in NIOSH, 2002). Benzo[a]pyrene has also been reported to have other adverse health effects including significant reproductive and developmental effects (ATSDR, 1996; Burns, Meade and Munson, 1996; Ramos, Chacob and Acosta Jr., 1996; Thomas, 1996; Rogers and Kavloc, 1996; WHO, 1998; NIOSH, 2002).

The focus of this mini-review was to summarize the adverse health effects associated with B[a]P inhalation. However, very little inhalation data were identified (IRIS, 1994; ATSDR, 1995; WHO, 1998; NIOSH 2002). Due to the potential carcinogenicity of B[a]P, its persistence in the environment, and the limited amounts inhalation data published, the adverse health effects reported via other routes of exposure were reviewed with an emphasis on the oral route. The primary literature sources for this assessment were the Agency for Toxic Substances and Disease Registry (ATSDR, 1995), the International Programme on Chemical Safety (WHO, 1998), Klaasen, Amdur and Doull (1996), and the National Institute for Occupational Safety and Health (NIOSH, 2002). Other documents describing PAH toxicity were available (*e.g.*, IARC, 1983; IRIS, 1994; EC 1994; MOE, 1997); however, the scope of this project did not allow for a detailed review of these documents.

4.1 Overview of Chemical Disposition

The chemical disposition of B[a]P is complicated and has been extensively documented. About 27 different metabolites have been identified, not all of which are toxic (Debriun cited in ATSDR, 1995; ATSDR, 1995; Gregus and Klaassen, 1996; Parkinson, 1996; IPSC, 1998). **In this report, due to the complexity of B[a]P metabolism in mammalian systems and the number of metabolites produced, only a *brief overview* of the absorption, distribution, metabolism, and excretion of B[a]P has been provided.**

Absorption of B[a]P depends mostly on the vehicle of administration and its lipophilicity. Although the extent of absorption is unknown, B[a]P is available via the respiratory tract; absorption depends on the animal species and the state B[a]P is presented (i.e. vehicle of administration) (ATSDR, 1995). In humans absorption via the gastrointestinal tract is low; in animals the rate of absorption after oral exposure depends on the animals species and the lipophilicity of the carrier vehicle (ATSDR, 1995). Dermal absorption occurs through passive

diffusion, and once again depends on the animal species and lipophilicity of the B[a]P preparation (ATSDR, 1995; WHO, 1998).

Studies of exposure to animals indicate that after oral or inhalation exposures, B[a]P is distributed to most tissues. No human distribution data was identified (ATSDR, 1995). Placental transfer has been reported to occur in animals; however, fetal levels are lower than maternal levels (ATSDR, 1995; Shendrikova and Aleksandrov, Shendrikova *et al.*, Takahashi, Baranova, Neubert and Tapken, cited in WHO, 1998). Lung tumours were reported to occur in mice and rabbit offspring (Beniashvili, Nikonova, cited in WHO, 1998).

The toxicity of benzo[a]pyrene (B[a]P) requires bioactivation in order to exert its toxicity. Metabolism appears to occur in most tissues via a number of pathways (ATSDR, 1995; Gregus and Klaassen, 1996; Parkinson, 1996; Pitot III and Dragon, 1996; Ramos, Chacon, and Acosta Jr., 1996; WHO, 1998). The metabolic products of B[a]P include: epoxide intermediates, dihydrodiols, phenols, quinines, and combinations of these products (ATSDR, 1995; Parkinson, 1996). Benzo[a]pyrene-7,8-hydrodiol-9,10-epoxide is B[a]P's ultimate toxicant[1]. It is an electrophilic epoxide produced after a two-step epoxidation process (cytochrome P450 enzymes and epoxide hydrolase) (Rogers and Kavlock, 1996; Parkinson, 1996). Benzo[a]pyrene-7,8-hydrodiol-9,10-epoxide is an electrophilic epoxide, and has been demonstrated to be highly mutagenic to mammalian cells and a much more potent tumourogen than B[a]P in mice lung (Parkinson, 1996). Details of the production of this and other metabolites of B[a]P have been studied through *in vivo* and *in vitro* studies (ATSDR, 1995. WHO, 1998, Klaasen, Amdur and Doull, 1996). There are a number of other factors (*e.g.*, diet, some holistic and pharmaceutical medicines, vehicle of administration), which have been reported to affect the metabolism and the carcinogenic potential of B[a]P (ATSDR, 1995).

Animal studies indicate the major route of excretion is in the feces. Little human excretion data was identified. In guinea pigs, excretion after dermal exposure has been reported to occur via urine as well as feces (ATSDR, 1995).

4.2 Genotoxicity and Carcinogenicity

Metabolism of the B[a]P into an epoxide (specifically benzo[a]pyrene-7,8-diol-9,10-epoxide) is required to produce the ultimate (mutagenic/carcinogenic) toxicant[1] (ATSDR, 1995; Parkinson, 1996; Pitot III and Dragan, 1996). Benzo[a]pyrene (B[a]P) has been well documented as genotoxic in many *in vivo* and *in vitro* studies (prokaryotic and mammalian cell assay systems) (US EPA a, cited in IRIS, 1994; ATSDR, 1995; IARC, cited in WHO, 1998; WHO, 1998; NIOSH, 2002). In fact, B[a]P has been used as a positive control in short-term mutagenic testing (WHO, 1998). Benzo[a]pyrene has been demonstrated to form DNA adducts in *in vivo* animal studies, as well as in a large number of *in vitro* test systems (of different animals and different cell types, including human) (WHO, 1998; NIOSH, 2002).

[1] Ultimate toxicant – the molecule responsible for the toxic effect(s) (Gregus and Klaassen, 1996).

There was insufficient human exposure data available for B[a]P to determine carcinogenic potential in humans (IRIS, 1994; WHO, 1998). Tests of over fifteen different animal species (from frogs to primates) have demonstrated B[a]P to be carcinogenic through many different routes (diet, gavage, inhalation, intratrachial instillation, intraperitoneal, intravenous, subcutaneous, and intrapulmonary injection, dermally, and transplacentally) (IRIS, 1994; WHO, 1998; NIOSH, 2002). Oral and dermal B[a]P exposures in experimental animals provides evidence of the chemical's carcinogenic potential (Albert *et al.*b, Berenblum and Haran, Cavaliere b, Chu and Malmgren, Habs *et al.*, Klein, Levin *et al.*, McCormick *et al.*, Neal and Rigdon, Rigdon and Neil, Snell and Stewart, Spamins *et al.*, Warshawsky and Barkley, Wattenberg and Leong, Wilson and Holland, Wynder and Hoffmann b, cited in ATSDR, 1995). Tumour location after exposure depends on the route of exposure (inhalation, ingestion, dermal, other), site of absorption, and the vehicle of administration (ATSDR, 1995, WHO, 1998). For example, dermal exposure produced skin tumours, inhalation and intratracheal instillation produced lung tumour, etc.. However, tumours have been observed to occur at sites distal to the route of exposure (*i.e.*, oral and subcutaneous exposures have produced lung tumours; intraperitoneal injection has produced liver tumours) (WHO, 1998).

Concurrent exposure to more than one PAH is common. Occupational exposure to mixtures of polyaromatic hydrocarbons (PAHs) (coke production, roofing, oil refining, coal gasification) indicates that PAHs are carcinogenic in humans (IRIS, 1994; ATSDR, 1995). Exposures to environmental mixtures (coal combustion effluent, vehicle exhaust, used motor lubricating oil, side stream tobacco smoke) are also thought to be carcinogenic due to the PAH component of the mixtures (IRIS, 1994; Grimmer *et al.*b., cited in WHO, 1998). The potential heath effects associated with this type of exposure depend on the mixture. Some studies have suggested that exposure to some of the weakly carcinogenic, or non-carcinogenic PAHs (*e.g.* benzo[e]pyrene, benzo[g,h,i]perylene, fluoranthene, pyrene) with B[a]P or other carcinogenic PAH, enhances the carcinogenic potential. The non-carcinogenic PAHs appear to have tumour-initiating and promoting properties (ATSDT, 1995). However, concurrent exposure to other non-carcinogenic PAHs (e.g., benzo[a]fluoranthene, benzo[k]fluoranthene, chrysene) has also been reported to lower the potential carcinogenic effects of B[a]P (ATSDR, 1995). Concurrent exposure to particulate matter and chemicals other than PAHs may also enhance the carcinogenic potential of a mixture (ATSDR, 1995). It is not within the scope of this report to examine effects associated PAH mixtures or associated with concurrent inhalation of a particulate or other chemicals which may occur in a PAH mixture (e.g., combustion gases, nitrates and nitrites).

Benzo[a]pyrene is classified by the EPA as a "B2 (probable human carcinogen)" (ATSDR, 1995; IRIS, 1994; WHO 1998). The American Conference of Governmental Industrial Hygenists (ACGIH) describes B[a]P as a "Suspected Human Carcinogen" (ACGIH, cited in NIOSH 2002). The International Agency for Research on Cancer (IARC) reports enough "Animal Sufficient Evidence" of carcinogenicity (IARC, 1983; IARC, cited in NIOSH, 2002) and classifies B[a]P in "Group 2A[2]" (probably carcinogenic to humans) (IARC, 1983; IARC, cited in NIOSH, 2002).

[2] This category is used when there is *limited evidence* of carcinogenicity in humans and *sufficient evidence* of carcinogenicity in experimental animals." (IARC, 1983).

4.3 Acute and Sub-Acute Effects

Acute effects usually occur rapidly as a result of short-term exposures, and are of short duration – generally for exposures less than 24 hours. Sub-acute effects usually occur as a result of exposures that are of an intermediate duration – generally for exposures lasting a few days to no greater than one month (Eaton and Klaasson, 1996).

4.3.1 Acute Human Effects

No studies of acute or sub-acute exposure in humans were identified for any route of exposure (IRIS, 1994; ATSDR, 1995; WHO, 1998; NIOSH, 2002).

4.3.2 Acute and Sub-Acute Animal Effects

A single sub-acute study of B[a]P inhalation in animals was identified (ATSDR, 1995; WHO, 1998). Exposure to 0.75 ppm B[a]P dust (7.7 mg B[a]P/m^3) for up to four weeks, produced no identifiable respiratory tract or kidney lesions (Wolff *et al.*, cited in ATSDR, 1995 and WHO, 1998).

Due to the persistence of B[a]P in the environment and the paucity of acute inhalation data, effects reported due to exposure via other routes was reviewed, with an emphasis on B[a]P ingestion. A few studies of acute exposures via other routes were identified, and indicate B[a]P to have moderate to low acute toxicity (WHO, 1998; NIOSH, 2002). Severity of B[a]Ps effects are dependent on dose, administration of dose (route and vehicle of administration), animal species, and animal genotype (ATSDR, 1995; WHO, 1998). Below is a summary of potential effects associated with acute and sub-acute B[a]P exposure in experimental animal studies.

4.3.2.1 Ingestion

Oral acute and sub-acute exposure to B[a]P in experimental animal studies has produced adverse reproductive and developmental effects (ATSDR, 1995). In addition, B[a]P was also demonstrated to have genotoxic and carcinogenic effects after acute and sub-acute oral exposures (ATSDR, 1995). Table 6 lists some examples of the lowest and highest NOAELs (No Observable Adverse Effect Levels) and LOAELs (Lowest Observable Adverse Effect Levels) reported in the literature for acute and sub-acute oral exposures.

Below is a summary of potential effects associated with acute and sub-acute B[a]P ingestion. Details regarding exposure concentrations, duration of exposure and animals species are included in Table 6.

Table 6 Examples of NOAELs and LOAELs Associated with Acute and Sub-Acute Benzo[a]pyrene Ingestion (Experimental Animals)

Effects Reported[a]	Exposure Period	Dose (mg B[a]P/kg body weight/day)	Species	Reference
Death:				
LD_{50}	Once	>1600	Mice	Awogi and Sato, cited in WHO, 1998.
Death due to mylotoxicity.	Daily for 1 – 4 wk	120	Mice (poor-affinity Ah receptor).	Legraverend, cited in WHO, 1998.
Mylotoxicty. NOAEL	Daily for up to 6 m	120	Mice (high-affinity Ah receptor).	Legraverend, cited in WHO, 1998.
Systemic:				
Gastrointestinal hepatic and renal systems. NOAEL.	1/d, 4 d	150 - gavage	Male rats.	Nousiainen *et al.*, ATSDR, 1994 and WHO, 1998.
Increased relative liver weights.	180 d	120	Mice	Robinson *et al.*, cited in WHO, 1998.
Multiple changes in enzyme levels (inhibition, induction, changes in blood or tissue levels).	Once	100	Rats.	FCTOD7 cited in NIOSH, 2002.
Reproductive and Developmental Effects:				
Reproductive. NOAEL.	19 – 21 d	133.3 – ad libitum.	Female mice	Rigeon and Neal, cited in ATSDR, 1995 and WHO, 1998.
Reduced pregnancy. NOAEL	10 d gd[b] 7-16	40 – gavage.	Female mice	Mackenzie and Angevine, cited in ATSDR, 1995.
Reduced pregnancy. Serious LOAEL	10 d gd[b] 7-16	160 – gavage.	Female mice	Mackenzie and Angevine, cited in ATSDR, 1995.
Fetal resorption. Serious LOAEL.	8d, gd 2 - 10	120 – in feed.	Female mice	Legraverend *et al.*, cited in ATSDR, 1995.
Reduced pup weight. NOAEL.	10 d gd[b] 7-16	10 – gavage.	Female mice	Mackenzie and Angevine, cited in ATSDR, 1995 and WHO, 1998.
Reduced pup weight at 20 days. Serious LOAEL.	10 d gd[b] 7-16	40 – gavage.	Female mice	Mackenzie and Angevine, cited in ATSDR,.
In F_1 pups a dose-response effect: impaired fertility, reduced testis weight and sterility in females.	10 d gd[b] 7-16	10, 40, 160	Mice	Mackenzie and Angevine, cited WHO, 1998.
Genotoxiciy:				

Effects Reported[a]	Exposure Period	Dose (mg B[a]P/kg body weight/day)	Species	Reference
B[a]P-DNA adducts in fetal and maternal tissues (fetal lung and liver; maternal liver and placenta).	Once on either gd 50, 100, or 150	5 – 50	Monkeys	Lu *et al.*, cited in ATSDR, 1995.
Gene mutations in mouse coat colour spot test.	Once	10	Mice	Davidson and Dawson, cited in ATSDR, 1995.
Dose-related increase in frequency of macronuclei in bone marrow cells.	Once	62.5 - 500	Mice	Awogi and Sato, cited in ATSDR, 1995.
Statistically significant increase in abnormal chromosomal morphology (in bone marrow cells).	Once	63	Male mice	Alder and Ingerwersen, cited in ATSDR, 1995.
Induced a clastogenic response in adult males, adult females, pregnant females, and fetal animals. (genetic damage was most severe in fetuses)	Once	150	Mice	Harper *et al.*, cited in ATSDR, 1995.
B[a]P-DNA adducts reported in liver, lung, and forestomach; linear dose response in liver and lung tissues.	21 days	6 doses: 0 – 6.5	Male mice	Culp and Beland, cited in ATSDR, 1995.
Carcinogenicity:				
Gastric neoplasms. NOAEL.	1-7 d	13.3 – ad libitum in feed.	Mice	Neal and Rigdon, cited in ATSDR, 1995.
Gastric neoplasms. Serious LOAEL.	1-7 d	33.3 – ad libitum in feed.	Mice	Neal and Rigdon, cited in ATSDR, 1995.
Increased incidence in forestomach and pulmonary tumours.	Once/w, 8 wk	1 – gavage	Mice	Wattenberg and Leong, cited in ATSDR, 1995.
77% incidence in mammary tumours (30% in controls) 90 weeks after exposure.	Once	100	Female rats	McCormick *et al.*, cited in ATSDR, 1995.
67% increased incidence mammary tumours.	Once/w, 8 wk	12.5	Female rats	McCormick *et al.*, cited in ATSDR, 1995.

[a] NOAEL, Less serious LOAEL, and Serious LOAEL as identified by ATSDR (1995).
[b] gd = gestational days.

4.3.2.1.1 Reproductive and Developmental Toxicity

Oral exposures to B[a]P resulted in reproductive and developmental toxicity in mice and possibly in rats (Mackenzie and Angevine, Rigdon and Neal, Rigdon and Rennels, Legraverend *et al.*, cited in ATSDR, 1995). Severity of the effects was dependent on dose and administration of dose (route and vehicle of administration), animal and strain of animal (ATSDR, 1995; WHO, 1998). Effects included sterility of progeny after maternal exposures, and reduced incidence of pregnancy in females, increased incidence fetal resorptions, reduced fetal body weight, and post-

partum death (Mackenzie and Angevine, Rigdon and Neal, cited in ATSDR, 1995 and WHO, 1998; Rogers and Kavlock, 1996). Adverse developmental effects have also been observed after intraperitoneal exposures (ATSDR, 1995).

4.3.2.1.2 Genotoxicity and Carcinogenicity

Intragastric exposures of intermediate duration (< 3 months) produced pulmonary adenomas, forestomach papillomas and increases in the incidence of forestomach and pulmonary tumours (Sparnins *et al.*, Wattenberg and Leong, cited in ATSDR, 1995). Acute and sub-acute exposures produced an increased incidence in mammary tumours in female rats (McCormick *et al.*, cited in ATSDR, 1995).

Consumption of a single B[a]P dose (33.3 mg/kg/day) was carcinogenic (forestomach neoplasms) in mice; exposure to a lower dose (13 mg/kg/day) did not result in a statistically significant increased incidence of neoplasms (Neal and Rigdon, cited in ATSDR, 1995).

4.3.2.2 Other Routes and Effects

Effects associated with dermal exposures (acute and sub-acute) have been reported in experimental animals studies (ATSDR, 1995; WHO, 1998). These effects include suppression of sebaceous glands, induction of melanocytes, contact hypersensitivities, and skin tumours (Bock and Mund, Iwata *et al.*, Klemme *et al.*, Old *et al.*, cited in ATSDR, 1995; WHO, 1998; Habs *et al.*, cited in WHO, 1998). Dermal exposure to pregnant mice was reported be associated with increased incidence of papillomas and carcinomas in all generations (Andrianova, cited in WHO, 1998).

Acute Intraperitoneal exposure of mice to B[a]P was reported to have adverse hematological effects (small spleen, marked cellular depletion and other pathological lesions), which resulted in death of the animals (Shubick and Porta, cited in ATSDR, 1995). Intraperitoneal injection of pregnant rat and mice produced immunological effects in the progeny (Csaba *et al.*, Csaba and Inczefi-Gonda, Urso and Johnsan cited in WHO, 1998).

Intraperitoneal and subcutaneous exposures have been reported to have immunosuppressive effects, effects on production of brain neurotransmitters (Blanton *et al.*, Dasgupta and Lahiri, Ginsberg *et al.*, Lyte and Bick, White and Holsapple, Wojdani and Alfred, Urso and Gengozian, Uros, Zhao *et al.*, cited in ATSDR, 1995; Deal, Parrott *et al.*, Schnizlein *et al.*, Xue *et al.*, cited in WHO, 1998), reproductive effects (Bui *et al.*, Cervello *et al.*, US EPA c, Hough *et al.*, Mackenzie and Angevine, Mattison *et al.*, Miller *et al.*, Swartz and Mattison, cited in ATSDR, 1995), developmental effects (Shum *et al.*, Payne, cited in ATSDR 1995), and to induce tumours (Bulay and Wattenberg, Soyka, cited in ATSDR, 1995; WHO, 1998).

In vitro studies using animal cells also reported immunotoxic effects (Ladies *et al.*, Ginsberg *et al.*, Lindemann and Park, cited in ATSDR, 1995). *In vitro* studies of B[a]P exposure to human placental tissues indicate that potential alterations in endocrine functions may occur (Bamea and Surtz-Swirski, Guyda *et al.*, cited in ATSDR, 1995).

4.4 Chronic Effects

4.4.1 Chronic Human Effects

Sub-chronic or intermediate exposures are generally one to three months; chronic effects occur as a result of long-term exposures and are of longer duration – generally as repeated exposures for more than 3 months (Eaton and Klaassen, 1996).

Few human studies of chronic inhalation exposures were identified (IRIS, 1994; ATSDR, 1995; WHO, 1998; NIOSH, 2002), and of those described, many were occupational exposures. There are a number of limitations to consider when using occupational data: i) the person(s) exposed generally is a healthy, young to middle aged, male adult; ii) concurrent exposures to other chemicals are very likely (in this case, particularly exposures to other PAHs, combustions gases and particulate matter); and, iii) the exposure concentrations are often difficult to define. Due to the fact that B[a]P is potential carcinogen and does not rapidly break down in the environment after precipitation from air sources, a short summary of sub-chronic and chronic effects after exposure via other routes is described below.

4.4.1.1 Inhalation

Table 7 lists some examples of the lowest and highest NOAELs (No Observable Adverse Effect Levels) and LOAELs (Lowest Observable Adverse Effect Levels) reported in the literature.

Below is a summary of potential effects associated with chronic inhalation of B[a]P. Details regarding exposure concentrations and duration of exposure are included in Table 7.

4.4.1.1.1 Respiratory

One study of exposure in the workplace to PAHs and specifically to B[a]P was reported by Gupta *et al.* (cited in ATSDR, 1995). The respiratory health of 667 workers was reported to be significantly affected. However, B[a]P exposure was concurrent with exposure to particulate matter, which of itself can produce respiratory problems and may exacerbate effects due to PAH exposure (ATSDR, 1995).

Table 7 Examples of NOAELs and LOAELs Associated with Sub-Chronic and Chronic Benzo[a]pyrene Inhalation (Human).

Effects Reported[a]	Exposure Period	Air Concentration [b] ppm (mg B[a]P/m^3)	Reference
Systemic:			
Reduced lung function, abnormal chest X-ray, cough, bloody vomit, throat and chest irritation. Serious LOAEL.	6 mo to < 6 yr	9.69×10^{-6} (0.0001)	Gupta *et al.*, cited in ATSDR, 1995.
Immunological and Lymphoreticular Effects:			
Reduced serum immunoglobins[c].	Average 15 yr	1.9×10^{-5} – 0.048 (0.0002 - 0.50)[c]	Szczeklik *et al.*, cited in ATSDR, 1995).
Genotoxic Effects:			
Positive results (PAH-DNA adducts in placentas and cord and placenta white blood cells) were reported in women exposed to PAHs from burning smoky coal indoors (with and without a chimney). LOAEL.	Not described.	0.037 (0.3832) - no chimney; 0.018 (0.184) - with chimney	Perera *et al.*, cited in ATSDR, 1995.
No cytogenic changes in peripheral lymphocytes. NOAEL.	Average 3.8 yr	1 mg PAH per 8-hour shift.	Becher *et al.*, cited in ATSDR, 1995.
Increased rate of mutations in peripheral lymphocyctes. LOAEL	2 – 46 yr	$\geq 8.7 \times 10^{-5}$ ($\geq$0.0005)	Perera *et al.*, cited in ATSDR, 1995.
No correlation between exposure concentrations and number of PAH-DNA adducts (in blood) and 1-hydroxypyrene (in urine).	Not described.	8.7×10^{-5} (0.0009)	Overebo *et al.*, Cited in ATSDR, 1995.
Increasing trend of PAH-DNA adducts with increasing occupational exposures (not statistically significant).	Not described.	1.9×10^{-7} – 5.8×10^{-6} (2.0×10^{-6} – 6×10^{-5})	Santella *et al.*, cited in ATSDR, 1995.
Carcinogenicity:			
High lung cancer rate associated with smoky coal use indoors without a chimney.	Not described.	Not described.	Mumford *et al.*, cited in ATSDR, 1995 and WHO, 1998.

[a] NOAEL, Less serious LOAEL, and Serious LOAEL as identified by ATSDR (1995).

[b] When both units of concentration were not provided in the literature, the following conversion factor and assumptions were used: mg/m^3 x 24.45/MW =ppm; MW=252.32, air at 25°C and 101.3 kPa (760mmHg) (Plog *et al.*, 1996).

[c] Concurrent exposure to other PAHs, sulphur dioxide, and carbon monoxide was reported. B[a]P exposure was used as a reference point (ATSDR, 1995). Smoking habits were not accounted for in this study.

4.4.1.1.2 Immunological and Lymphoreticular Effects

Humoral suppression has been reported in worker at a foundry (Szczelik *et al.*, cited in ATSDR, 1995). *In vitro* studies with animals cells have reported that immunosuppressive effects occur after exposures to carcinogenic PAHs; suppression was not reported to occur in non-carcinogenic PAH studies (Blanton *et al.*, Lyte and Bick, White and Holsapple, Hahon and Booth, Urso *et al.*, cited in ATSDR, 1995).

4.4.1.1.3 Carcinogenicity

The US EPA (IRIS, 1994), ATSDR (1995) and WHO (1998) did not identify any adequate human inhalation studies examining the potential carcinogenicity of B[a]P alone. However, increased mortality from lung cancer has been reported in workers exposed to B[a]P concurrent with other PAHs and other products of combustion (Lloyd, Mazumdar *et al.*, Redmond *et al.*, Hammond *et al.*, Maclure and MacMahon, Wynder and Hoffman, cited in ATSDR, 1995). ATSDR (1995) reports these studies to provide qualitative evidence that B[a]P may be a potential carcinogen.

4.4.1.2 Ingestion

No human studies of acute or sub-acute oral B[a]P exposure were identified (ATSDR, 1995).

4.4.1.3 Other Routes and Effects

Few human studies of dermal B[a]P exposure were identified (ATSDR, 1995). Polyaromatic hydrocarbon (PAH) mixtures were sometimes used to treat specific skin disorders. Adverse effects (such as warts, skin eruptions) have been reported in humans after sub-chronic dermal applications of a 1% B[a]P solution (Cottini and Mazzone, cited in ATSDR, 1995).

4.4.2 Sub-Chronic and Chronic Animal Effects

Few animal studies of sub-chronic and chronic inhalation were identified (IRIS, 1994; ATSDR, 1995; WHO, 1998). Due to the fact that B[a]P is potential carcinogen and does not rapidly break down in the environment after precipitation from air sources, a short summary of sub-chronic and chronic effects after exposure via other routes is described below with an emphasis on ingestion. The majority of the studies identified were carcinogenicity studies (WHO, 1998). Severity of B[a]Ps effects is dependent on dose and administration of dose (route and vehicle of administration), animal species, and animal genotype (ATSDR, 1995; WHO, 1998).

4.4.2.1.1 Inhalation

Table 8 lists some examples of the lowest and highest NOAELs (No Observable Adverse Effect Levels) and LOAELs (Lowest Observable Adverse Effect Levels) reported in the literature for sub-chronic and chronic exposures.

Table 8 Examples of NOAELs and LOAELs Associated with Sub-Chronic and Chronic Benzo[a]pyrene Inhalation (Experimental Animals)

Effects Reported[a]	Exposure Period	Air Concentration [b] ppm (mg B[a]P/m^3)	Species	Reference
Death:				
Dose –related decrease in survival after 60 weeks.	109 wk	4.5 (46.5)	Hamster	Thyssen *et al.*, cited in ATSDR, 1995.
Carcinogenicity:				
Lung tumours. NOAEL.	4.5 h/d 5 d/wk 16 wk	4.34 (44.8)	Hamster	Thyssen *et al.*, cited in WHO, 1998.
Dose-response: tumours in the nasal cavity, pharynx, larynx, and trachea (no lung tumours).	109 wk	0.92 or 4.51 (9.5 or 46.5)	Hamster	Thyssen *et al.*, cited in WHO, 1998.
Increase in respiratory tract tumours and neoplasms of the upper digestive tracts. Serious LOAEL.	3-4.5 h/d 7 d/wk 109 wk	0.92 (9.5)	Hamster	Thyssen *et al.*, cited in ATSDR, 1995.

[a] NOAEL, Less serious LOAEL, and Serious LOAEL as identified by ATSDR (1995).

[b] When both units of concentration were not provided in the literature, the following conversion factor and assumptions were used: mg/m^3 x 24.45/MW =ppm; MW=252.32, air at 25°C and 101.3 kPa (760mmHg) (Plog *et al.*, 1996).

Below is a summary of potential effects associated with sub-chronic and chronic B[a]P inhalation. Details regarding exposure concentrations, duration of exposure and animal species examined are included in Table 8.

4.4.2.1.2 Death

In hamsters, decreased rate of survival (possibly due to tumours of the larynx and pharynx interfering with food intake) was reported in a chronic inhalation study (Thyssen *et al.*, cited in ATSDR, 1995).

4.4.2.1.3 Carcinogenicity

Chronic inhalation of B[a]P has been reported to induce tumours in the respiratory tract (nasal, pharynx, and larynx) in mice and hamsters (Shulte *et al.*, Thyssen *et al.*, cited in ATSDR, 1995).

4.4.2.2 Ingestion

Table 9 lists some examples of the lowest and highest NOAELs (No Observable Adverse Effect Levels) and LOAELs (Lowest Observable Adverse Effect Levels) reported in the literature for sub-chronic and chronic exposures.

Table 9 Examples of NOAELs and LOAELs Associated with Sub-Chronic Benzo[a]pyrene Ingestion (Experimental Animals)

Effects Reported[a]	Exposure Period	Dose (mg B[a]P/m^3)	Species	Reference
Systemic Effects:				
Increased liver weight. Less serious LOAEL.	6 mo	120	Female Mice	Robinson *et al.*, cited in ATSDR, 1995.
Aplastic anemia. Serious LOAEL	6 mo	120	Female Mice	Robinson *et al.*, cited in ATSDR, 1995.
Carcinogenicity:				
NOAEL.	30 – 197 d	1.3 – ad libitum	Female mice	Neal and Rigdon, cited in ATSDR, 1995.
Gastric tumour. Serious LOAEL.	30 – 197 d	2.6 – ad libitum	Female mice	Neal and Rigdon, cited in ATSDR, 1995.
Increased pulmonary tumours.	30 weeks	3 mg/animal	Mice	Wattenberg and Leong cited in WHO, 1998.
Papillomas; squamous cell carcinomas. Serious LOAEL.	23 – 238 d	33.3 – ad libitum	Female mice	Rigdon and Neal, cited in ATSDR, 1995.
Tumours of the forestomach (68/108). Serious LOAEL.	80 – 140 d	33.3 – ad libitum	Female mice	Rigdon and Neal, cited in ATSDR, 1995.
37% developed Leukemia. Serious LOAEL.	"intermediate"	33.3 – ad libitum in feed.	Mice	Rigdon and Neal, cited in ATSDR, 1995.

[a] NOAEL, Less serious LOAEL, and Serious LOAEL as identified by ATSDR (1995).

Below is a summary of potential effects associated with sub-chronic and chronic B[a]P ingestion. Details regarding exposure concentrations, duration of exposure and animal species examined are included in Table 9.

4.4.2.2.1 Carcinogenicity

Sub-chronic exposures in mice produced pulmonary adenomas and forestomach papillomas; increased incidence of forestomach and pulmonary tumours; and, increased incidence of leukemia (Sparnins *et al.*, Rigdon and Neal, Rigdon and Neal cited in ATSDR, 1995). A chronic

oral exposure to B[a]P has been associated with a statistically significant increase in number of tumours (stomach, forestomach) (ATSDR a, cited in WHO, 1998).

4.4.2.3 Other Routes and Effects

IARC (cited in WHO, 1998) reported B[a]P to produce tumours after dermal, subcutaneous, and/or intramuscular exposures (also reported in ATSDR, 1995). Severe, long lasting metaplasic changes were observed in rat trachea after pellets of B[a]P were implanted for four weeks (Topping *et al.*, cited in ATSDR, 1995).

Hypersensitivity was reported in two animal studies after injection of a B[a]P solution (Olds *et al.*, Klemme *et al.*, cited in WHO, 1998).

4.5 Other Effects

Benzo[a]pyrene is also a smooth muscle toxin altering smooth muscle cell proliferation through several mechanisms. It may act as a promoter the atherogenic process, resulting in atherosclerosis in experimental animals (Hough *et al.*, Penn and Snyder, Pessah-Rasmussen, cited in ATSDR, 1995; Ramos, Chacon and Acosta, 1996). *In vitro* studies of human cell cultures also indicated B[a]P may have vascular toxicity (Pessah-Rasmussen, cited in ATSDR, 1995). Benzo[a]pyrene has also been reported to have phototoxic effects with exposure to ultraviolet light following B[a]P exposure. The effects observed included: haemolysis of human erythrocytes; inactivation of *Escherichia coli*; and, enhancement of the tumourogenic response in mice (Kagan *et al.*, Gensler *et al.*, cited in WHO, 1998).

Polyaromatic hydrocarbons (PAHs) have been documented to be potent immunosupressents through suppression of the antibody response (T-cell dependant and independent antibodies) (Burns, Meade, and Munson, 1996). Inhalation of PAH mixtures has been reported to increase incidence of tumours and increase mortality (Schulte *et al.* cited in ATSDR, 1995; Heinrich, Heinrich *et al.*, a,b, cited in WHO, 1998). Ingestion studies of PAH mixtures also indicated carcinogenicity (Culp *et al.*, cited in WHO, 1998).

4.6 Summary Of Adverse Health Effects Of Benzo[a]Pyrene

The primary end points of toxicity associated with benzo[a]pyrene (B[a]P) exposure are carcinogenicity, reproductive toxicity, and developmental toxicity (ATSDR, 1995; Casarrett and Doull, 1996; WHO, 1998; NIOSH, 2001). Other non-carcinogenic effects also occur at doses know to be carcinogenic (growth inhibition, Immunosuppression, liver damage, and irritation/hypersensitivities) (ATSDR, 1995; WHO, 1998). Severity of the effects depends on dose, administration of dose (route and vehicle of administration), and animal species, age and genotype (ATSDR, 1995; WHO, 1998).

Benzo[a]pyrene is classified by the US EPA as a "B2 (probable human carcinogen)" (ATSDR, 1995; IRIS, 1994; WHO 1998; NIOSH, 2002). The American Conference of Governmental Industrial Hygenists (ACGIH) describes B[a]P as a "Suspected Human Carcinogen" (ACGIH, cited in NIOSH 2002). The International Agency for Research on Cancer (IARC) reports enough "Animal Sufficient Evidence" of carcinogenicity (IARC, cited in NIOSH, 2002) and classifies B[a]P as a "Group 2A" carcinogen (probably carcinogenic to humans) (IARC, 1983; IARC cited in NIOSH, 2002).

5.0 EFFECTS ON VEGETATION AN MATERIALS

5.1 Vegetation

The biological effects of PAHs have been reviewed by Edwards (1983). Plants acquire PAHs by absorption through their roots. Once entry has occurred, it is thought that PAHs can then be translocated to all plant tissues. In addition, PAHs can also enter the plant by foliar deposition. The following paragraphs provide the results of a literature review that was conducted on the effects of PAHs on vegetation, with specific emphasis on benzo[a]pyrene (B[a]P).

Currently the knowledge of uptake and translocation is limited in that only a relative small amount of data are present for B[a]P. Physiological studies of uptake, translocation and accumulation of B[a]P have been conducted on a number of plant species such as rye, wheat, maize, potatoes, lettuce, carrots, green beans, radishes and corn (reviewed by Edwards, 1983), in a number of different medias such as nutrient solutions, sand and soil. Edwards (1983) concluded that terrestrial plants can take up PAHs, including B[a]P, through both their roots and/or leaves and translocate it to various plant tissues. The uptake and translocation rates vary amongst plant species. In addition, other sources of variation in uptake can be attributed to a number of factors such as concentration, the phase of B[a]P (vapour or particulate) and the media supporting the plant (hydroponic solution, sand or soil).

PAHs are common contaminants of terrestrial and aquatic ecosystems. Toxicological studies for defining the potential hazards of PAHs have been conducted in the absence of UV radiation. Currently, evidence has indicated that coexposure of UV light and near UV light and the specific PAH can result in increased toxicity (Arfsten *et al.*, 1996). Light dependent toxicity has been evaluated for anthracene, pyrene, benzo[a]pyrene, fluoanthene, phenanthrene, and naphthalene (Mallakin *et al.*, 2002; Ren *et al.*, 1993 and 1996; Huang *et al.*, 1993; 1995; 1996).

In summary, B[a]P has been demonstrated to exhibit phototoxicity. In algae, Cody *et al.* (1984) have shown experimental evidence that suggested B[a]P is phototoxic to aquatic flora. B[a]P and a number of B[a]P metabolites (3,6-quinone, 6-hydroxy, 9-hydroxy, 3-hydroxy and 1,6-quinone) at a concentration 40 µg/L inhibit the growth rate of *Selenastrum capriconutum* populations when treated under cool white lights (<500 nm) for a duration of 4 to 7 days, whereas after treatment under gold lights, no inhibition was observed at these levels. The EC_{50} for B[a]P under white light was 0.175 µmol/L, whereas the EC_{50} under black florescent lighting was an order of magnitude lower (0.010 µmol/L). In the aquatic higher plant *Lemna gibba* (Duckweed), coexposure of benzo[a]pyrene, anthracene, phenanthrene and their respective photoproducts resulted in inhibition of growth and chlorosis (Huang *et al.*, 1993). Under simulated sunlight radiation minimal growth (<20%) inhibition was observed for B[a]P at 2 µg/ml.

In addition, it is thought that flora, both aquatic and terrestrial, can bioconcentrate PAHs. The algae *Oedogonium cardicacum*, after a 3 day exposure to B[a]P, had a bioconcentration factor

(BCF) of 5258 (Lu *et al.*, 1977). In a higher plant (canola), Ren *et al.* (1996) studied the effect of B[a]P and photomodified B[a]P on germination and early seedling growth. They found that toxicity of B[a]P to canola demonstrated a log-linear response with an EC_{50} of 1-4 mg/L, when present in the liquid growth media. Photomodified B[a]P however had an EC_{50} range of one-half that of B[a]P. Germination was unaffected by either B[a]P or photomodified B[a]P, when the B[a]P was present in the germination media.

In canola and cucumber, Huang *et al.* (1996) demonstrated that foliar application of B[a]P (treatment of 2 mg/L, 4 times daily for 20 days), resulted in a decrease in shoot biomass of 55 % of the untreated controls. When the concentration was increased to 8 mg/L of B[a]P, shoot biomass decreased to 35% of the untreated controls. Photomodified B[a]P inhibited biomass accumulation in the same manner as B[a]P. Treatment with B[a]P caused chlorosis of the leaves, in contrast, photomodified B[a]P treatment did not. In addition, photosynthetic activity decreased within 2 hr of B[a]P exposure and after 24 hours, the toxic effects were more evident in the plants. Huang *et al.* (1996) attributed dramatic shoot biomass decrease in comparison to root biomass to the site of exposure (foliar) and photomodification of B[a]P. Huang *et al.* (1996) proposed that B[a]P and its photoproducts can depress both cell division and cell expansion. These results indicate that B[a]P in rain and surface waters could be a hazard to terrestrial plants.

A number of articles have dealt with PAH levels in varying plant species (reviewed by Edwards, 1983). Edwards (1983) expressed the following tentative conclusions: (i) vegetation near PAH sources have a higher PAH load in comparison to distant counterparts, (ii) PAH levels are higher on plant surfaces, (iii) generally the above-ground tissue of the plant has a higher PAH concentration than the below-ground tissue, and (iv) a possible correlation exists between leaf surface area and absorption from the atmosphere. Howsam *et al.* (2000) found that a number of PAHs were present in the leaves of oak, ash and hazel. B[a]P was responsible for approximately 4.2 % of PAHs load in these plant tissues.

Lodovici *et al.* (1994) evaluated the levels of 10 PAHs of the bay evergreen tree (*Laurus nobilis*) located in 15 sites in the Arno valley, near Florence, Italy. Lodovici *et al.* (1994) found that B[a]P levels varied from 2.5 to 22.5 $\mu g\ kg^{-1}$ (dry weight). Kipopoulou *et al.* (1999) determined the PAH content of vegetables produced near industrial areas of Thessalonki, Greece. Kipopoulou *et al.* (1999) analyzed plant tissues from cabbage, carrots, leek, lettuce and endive for PAHs and found that, for B[a]P, concentrations in the inner tissue of the plants were in the range of 0.08-0.74 $\mu g\ kg^{-1}$ (dry weight). In addition, the Kipopoulou *et al.* (1999) monitored ambient air levels of PAHs and found that these levels correlated with the levels present in the inner tissue of the plants.

5.2 Materials

Benzo[a]pyrene will normally exist as a mixture with other PAHs in the atmosphere and be associated with airborne particulate matter at ambient temperatures. Benzo[a]pyrene, per se, is not of concern with respect to material effects. However, airborne particles laden with PAH mixtures may – in part – contribute to deposition onto surfaces causing soiling. In addition, particles deposited on a surface can adsorb or absorb acidic gases (e.g. SO_2 and NO_2), thus

serving as nucleation sites for these acidic gases (Baedecker *et al.*, 1991). This may accelerate physical and chemical degradation of material surfaces that normally occur when materials are exposed to environmental factors such as wind, sun, temperature fluctuations, and moisture. Haynie and Lemmons (1990) describe soiling as the contrast in reflectance of particles on a substrate compared to the reflectance of a bare substrate. Soiling of materials is a concern because it results in more frequent cleaning and repainting, thereby reducing its lifetime usefulness and increasing costs associated with maintenance of the materials.

Haynie (1986) reported that it is difficult to determine the amount of deposited particles that cause an increase in soiling. However, Haynie (1986) indicated that soiling is dependent on the particle concentration in the ambient environment, particle size distribution, and the deposition rate and the horizontal or vertical orientation and texture of the surface being exposed. Schwar (1998) reported that the buildup of particles on a horizontal surface is counterbalanced by an equal and opposite depletion process. The depletion process is based on the scouring and washing effect of wind.

6.0 AIR SAMPLING AND ANALYTICAL METHODS

6.1 Reference Methods

Air sampling and analytical methods for benzo[a]pyrene used in practice by established agencies are reported. The most frequently used technique for sampling benzo[a]pyrene is to use a high-volume air sampler equipped with a filter for trapping the particle-bound portion from the air and a solid adsorbent for collecting the portion in the vapour phase. Widely employed and accepted reference air monitoring methods for benzo[a]pyrene based on this concept have been developed, tested and reported by the United States Environmental Protection Agency (US EPA) and National Institute of Occupational Safety and Health (NIOSH). Refer to Table 10 for a description of individual method advantages and disadvantages.

6.1.1 US EPA Compendium Method TO-13A

US EPA has developed a methodology suitable for sampling ambient air for trace-level concentrations of benzo[a]pyrene. US EPA Compendium Method TO-13A describes the determination of individual polycyclic aromatic hydrocarbons (PAHs) (including benzo[a]pyrene) in ambient air using a combination filter and sorbent cartridge sampling system with subsequent analysis by gas chromatography/mass spectrometry (GC/MS) (US EPA, 1999).

In this method, a high-volume pump is used to draw ambient air through a glass-fibre filter and either a polyurethane foam (PUF) or XAD-2 adsorbent cartridge (in the case of benzo[a]pyrene, PUF is generally used). The high volume pump is operated for 24 hours at a rate of 0.23 m^3/min to collect a total volume of 325 m^3. After sampling, the filter and cartridge are extracted using 10% diethyl ether. The extract is then concentrated using the Kuderna-Danish technique, diluted, and cleaned using column chromatography. The cleaned extract is analyzed by GC/MS. The detection limit for the overall procedure is 0.5 to 500 ng/m^3.

Advantages of this method include: allows for sample dilution if concentration is too high during analysis; repeated analysis is possible; high volume sampling provides lower detection limits; and filter and sorbent are low cost. Disadvantages of this method include: interferences due to contamination of solvents, reagents, glassware and sampling hardware; coeluting contaminants may cause interference with target analytes; and heat, ozone, NO_2, and ultraviolet light may cause sample degradation.

6.1.2 NIOSH Method 5506

In addition to the air monitoring method for benzo[a]pyrene developed by the US EPA, the NIOSH has also developed methods for benzo[a]pyrene that are suitable for occupational, personal and area monitoring. The first methodology used by the NIOSH to determine

benzo[a]pyrene in air (NIOSH Method 5506) consists of collecting benzo[a]pyrene through a filter and sorbent tube with subsequent chemical analysis by high performance liquid chromatography (HPLC) followed by fluorescence detection (NIOSH, 1998). Advantages of this method are that it is suitable for peak, ceiling and TWA determinations of polynuclear aromatic hydrocarbons and it may be used for simultaneous measurements. Disadvantages include possible interferences from any compound that elutes at the same HPLC retention time; heat, ozone, NO_2, or UV light may cause sample degradation; and it is not applicable for samples that contain large amounts of highly adsorptive particulate matter. Sampling is conducted by drawing air through a PTFE filter and solid sorbent tube (XAD-2, 100 mg in the front section and 50 mg in the back section) using a personal sampling pump. The suggested flow rate is 2.0 L/min and the volume collected should be between 200 L and 1000 L. The contents of the tube are extracted with acetonitrile and analyzed by HPLC with fluorescence detection. The level of detection for concentrations of benzo[a]pyrene using this method is 0.1 µg/sample.

6.1.3 NIOSH Method 5515

The final methodology used by the NIOSH to determine benzo[a]pyrene in air (NIOSH Method 5515) consists of collecting benzo[a]pyrene through a filter and sorbent tube with subsequent chemical analysis by capillary column GC with flame ionization detection (FID) (NIOSH, 1994). Advantages of this method are that it is suitable for peak, ceiling and TWA determinations of polynuclear aromatic hydrocarbons and it may be used for simultaneous measurements. Disadvantages include possible interferences from any compound that elutes at the same HPLC retention time and heat, ozone, NO_2, or UV light may cause sample degradation. Sampling is conducted by drawing air through a PTFE filter and solid sorbent tube (XAD-2, 100 mg in the front section and 50 mg in the back section) using a personal sampling pump. The suggested flow rate is 2.0 L/min and the volume collected should be between 200 L and 1000 L. The contents of the tube are extracted with an appropriate solvent and analyzed by GC/FID. The working range for benzo[a]pyrene with this method is 3 to 150 µg/m^3 for a 400-L air sample. The level of detection for concentrations of benzo[a]pyrene using this method is 0.5 µg/sample.

6.2 Alternative, Emerging Techniques

Reports, journal articles, conference proceedings and other sources known to contain information on ambient measurement methods for chemicals such as benzo[a]pyrene were reviewed to determine the current status of alternative and emerging techniques. The result of the review indicates several types of alternative and emerging techniques have been developed and reported. In general, most non-standard methods are variations or modifications of the reference methods previously mentioned. However, several unique emerging methods and technologies have been described.

Table 10 Method Advantages and Disadvantages

Method	Advantages	Disadvantages
US EPA TO-13A	Allows for sample dilution if concentration is too high during analysis Repeated analysis is possible High-volume sampling provides lower detection limits Filter and sorbent are low cost	Method has interferences due to contamination of solvents, reagents, glassware and sampling hardware Coeluting contaminants may cause interference with target analytes Heat, ozone, NO_2, and ultraviolet light may cause sample degradation
NIOSH Method 1506	Suitable for peak, ceiling and TWA determinations of polynuclear aromatic hydrocarbons It may be used for simultaneous measurements	Possible interferences from any compound that elutes at the same HPLC retention time Heat, ozone, NO_2, or UV light may cause sample degradation Not applicable for samples that contain large amounts of highly adsorptive particulate matter
NIOSH Method 5515	Suitable for peak, ceiling and TWA determinations of polynuclear aromatic hydrocarbons It may be used for simultaneous measurements	Possible interferences from any compound that elutes at the same HPLC retention time Heat, ozone, NO_2, or UV light may cause sample degradation

6.2.1 Alternative Techniques

As with the reference methods described earlier, the principle stages of alternative techniques for ambient monitoring of polycyclic aromatic hydrocarbons (PAHs) such as benzo[a]pyrene involve sampling, extraction, cleanup and analysis.

6.2.1.1 Sampling

The sampling step is by far the most important source of variability when investigating ambient PAH concentrations. The physical state of the PAH of interest in the atmosphere must be considered when selecting the sampling technique. It has been shown that compounds with five or more rings (such as benzo[a]pyrene) are almost exclusively absorbed on airborne particles, whereas lower molecular mass PAHs are partially or totally present in the vapour phase (WHO, 1998). However, since ambient conditions such as season, meteorology, time of day, and temperature can alter the amount of the PAH found in each phase, collection methods that use either a filtration system (CASA, 2004) or adsorbent alone may grossly underestimate ambient PAH concentrations. Therefore, the most common method for sampling ambient PAH is to use an active air sampler which has been equipped with a filter for trapping the particles from the air and a solid adsorbent for collecting substances in the vapour phase.

A high-volume air sampler (0.3 to 1.5 m^3/min) operated for 24 hours or longer (to avoid sample degradation and losses) is the most frequently used instrument for sampling PAH. Low-volume air samplers (0.001 to 0.3 m^3/min) have been tested recently (ARPEL, 1998). The advantage of this type of sampler is the high sampling capacity and possible high time resolution. However,

the small sample volumes place high demands on the analytical methods. The choice of sampling rate ultimately depends upon the integration period, ambient concentrations and analytical sensitivity.

Most studies have sought to determine PAH concentrations from total suspended particles (TSP) (CASA, 2004; Environment Canada, 1998). However, some studies have used size-selective inlets to collect particulate matter with aerodynamic diameters of 10 µm or less (PM_{10}) (WHO, 1998; Peltonen and Kuljukka, 1995).

Two types of filters, glass-fibre and silver membrane, have been utilized most often for sampling airborne particulate PAH with both high-volume and low-volume samplers. Other filter media that can be used for sampling include quartz fibres, polytetrafluoroethylene (PTFE) and PTFE-coated glass-fibre (WHO, 1998). The glass-fibre filter is the popular choice for airborne particulate matter sampling because it has good mechanical strength and is not expensive.

Several attempts have been made to collect volatile PAH fractions including impregnating the filter medium, using a back-up filter or a solid adsorbent behind the filter (WHO, 1999). Employing appropriate solid absorbents downstream of the filter appears to be the most practical vapour collection technique. Vapour-phase PAHs are normally trapped onto plugs of polyurethane foam (PUF) or XAD-2 located behind the particulate filter (ARPEL, 1998; Hawthorne et al., 1989). Other absorbents that have been successfully used to trap PAHs include Porapak PS and Tenax GC, Tenax TA, Tenax GR and C18 (WHO, 1998).

PUF and XAD-2, while being the most common adsorbents, have their inherent weaknesses and strengths. For example, although most adsorbents have shown high collection efficiencies for benzo[a]pyrene, only XAD-2 has high collection efficiency for volatile PAHs such as naphthalene. PUF cartridges are easier to handle in the field and demonstrate superior flow characteristics during sampling. PUF cartridges can also be used to collect and analyze for other hazardous air pollutants such as organochlorine pesticides and polychlorinated biphenyls.

Some recommend the use of both adsorbents, together, to address post-collection volatilization problems associated with volatile and other reactive PAHs (DEH, 1999). It is recommended that XAD-2 be used sandwiched between two PUF plugs. Since XAD-2 can retain up to 99% naphthalene, the most volatile PAH, this configuration should be used with the filter paper for collecting PAHs in ambient air.

6.2.1.2 Extraction

The PAHs collected onto the filters or adsorbed onto solid adsorbents must be extracted with appropriate methods prior to analysis. Traditionally, PAHs are extracted by Soxhlet extraction using an appropriate solvent such as acetone, n-hexane, toluene, benzene or dichloromethane (or combinations of the solvents) (WHO, 1999). The Soxhlet method is very efficient for extracting PAHs and it is the preferred procedure in the US EPA method TO-13A for PAH determination (US EPA, 1999). Unfortunately, the Soxhlet extraction method suffers from disadvantages such

as requiring extremely long extraction times and the use of a large volume of hazardous solvents (Peltonen and Kuljukka, 1995).

As an alternative to Soxhlet extraction, ultrasonication is gaining widespread acceptance due to lower solvent volume requirements, superior recovery efficiencies, reduced extraction times and temperatures, and possibly reduced sample degradation (DEH, 1999). Supercritical fluid extraction (SFE) with carbon dioxide in the presence of a modifier (such as 10% methanol) is preferable to the conventional extraction methods because SFE is much less time consuming and has comparable or better PAH extraction recovery (Hawthorne et al., 1989). Other potential methods that seem to be feasible alternatives include focused microwave extraction (FME), sublimation, direct extraction and thermal desorption (ARPEL, 1998; Peltonen and Kuljukka, 1995).

6.2.1.3 Cleanup

After extraction, samples must be purified (cleaned) to remove unwanted contaminants that could interfere with subsequent analytical procedures. Extracted samples are normally purified by column chromatography using a variety of column sorbents such as alumina, silica gel, C_{18}, Sephadex or Florisil (DEH, 1999). High performance liquid chromatography (HPLC) can also be used for the cleanup of PAHs in sample extract (Peltonen and Kuljukka, 1995). Thin layer chromatography (TLC) on glass plates is yet another technique that has been used successfully on ambient air samples (WHO, 1999). Conventional chromatographic column techniques may be substituted by eluting extracted samples through prepacked cartridges, which have the advantage of using less time and solvent (WHO, 1998).

6.2.1.4 Analysis

A wide variety of analytical methods have been used for determining trace concentrations of PAHs in environmental samples. The most common of these include gas chromatography (GC) using various detectors, HPLC using various detectors and TLC with fluorimetric detection. Each technique has a number of relative advantages, however, they are expensive and require qualified operating personnel. Nevertheless, they are considered necessary in order to analyze samples with accuracy and precision. The detection limits of these instruments for PAHs, as reported in some studies and cited in IPCS monogram, are in the ng/m^3 ambient concentration range (WHO, 1998).

Chromatographic analysis methods have been used to give high resolution among different PAHs. Various detection devices used for GC quantification include flame ionization detector (FID), mass spectrometry (MS), photoionization detector (PID), electron capture detector (ECD), infrared absorption detector (IAD), Fourier transform infrared spectrometer (FTIR), laser induced molecular fluorescence detector (LIMF), diode array detector (DAD), nitrogen-phosphorous detector (NPD), and gas phase fluorescence detector (GPFDA) (DEH, 1999; ARPEL, 1998; WHO, 1998; and Peltonen and Kuljukka, 1995). These detectors exhibit widely different sensitivity, specificity, and ability to identify compounds.

Compared to GC, HPLC is generally less suitable for separating samples containing complex PAH mixtures (DEH, 1999). However, the ultraviolet (UV) or fluorescence detectors used in HPLC are highly specific and sensitive and can resolve the peaks of various PAHs that cannot be adequately resolved by GC. The detection limit of the fluorescence detector is an order of magnitude greater than the UV detector. Another advantage of the fluorescence detector is that it can determine PAHs in the presence of other non-fluorescent compounds.

TLC or high performance thin layer chromatography (HPTLC) with fluorimetric detection are commonly used only for identifying individual compounds during screening or for identifying selected PAHs of interest. It is an inexpensive, quick analytical technique but has low separation efficiency (Peltonen and Kuljukka, 1995).

A number of less commonly used analytical techniques are available for determining PAHs. These include synchronous luminescence spectroscopy (SLS), resonant (R)/nonresonant (NR)-synchronous scan luminescence (SSL) spectrometry, room temperature phosphorescence (RTP), ultravioletresonance Raman spectroscopy (UV-RRS), x-ray excited optical luminescence spectroscopy (XEOL), laser-induced molecular fluorescence (LIMF), supersonic jet/laser induced fluorescence (SSJ/LIF), low-temperature fluorescence spectroscopy (LTFS), high-resolution low-temperature spectrofluorometry, low-temperature molecular luminescence spectrometry (LT-MLS), and supersonic jet spectroscopy/capillary supercritical fluid chromatography (SJS/SFC) (WHO, 1998).

6.2.2 Emerging Techniques

The current methods for the sampling and analysis of PAHs are labour-intensive and complicated, requiring skilled personnel. To overcome these difficulties, a number of emerging technologies have been developed, including: annular denuder methods, super critical fluid extraction (SFE) methods, electrophoresis methods, continuous PAH analyzers, and passive samplers.

Various combinations of annual diffusion denuder arrangements have been employed in PAH sampling. The use of annular diffusion denuders with back-up filters appears to have the potential to reduce artifact formation during PAH sampling (DEH, 1999). However, the denuder coating limits the success of this method. Presently available coatings can only be used at slow sample flow rates. New developments including gas-and-particle (GAP) samplers based on annular diffusion denuder techniques have not gained wide acceptance, but their development continues (ARPEL, 1998).

SFE can be coupled directly to capillary GC/MS systems to determine PAHs, considerably reducing sample handling and expediting measurements. Shimmo *et al.* (2002) have developed an on-line supercritical fluid extraction-liquid chromatography-gas chromatography-mass spectrometry (SFE-LC-GC-MS) system for the analysis of particulate PAHs. The analysis can be carried out in a closed system without tedious manual sample pretreatment and with no risk of errors by contamination of loss of the analytes. The limits of detection for filter samples with this technique varied from 0.02 to 0.04 ng/m^3 for a 24-hour sampling period.

Capillary electrophoresis (CE) methods have emerged as important tools in chemical analysis and have the potential to rival traditional separation techniques such as GC and HPLC. These techniques involve the use of electroosmotic flow (EOF), an electrically induced force that moves a mobile phase through a packed bed column, to effect separation of different electrical species on the column.

One CE method that has been successfully applied to ambient PAH samples is micellar electrokinetic capillary chromatography (MEKC) with UV-laser excited fluorescence detection (DEH, 1999). In this method, electrically neutral PAHs are given a charge by the addition of an ionic surfactant to a buffer containing the PAHs. The surfactants form micelles that react with the PAHs. Separation of the PAHs is based on their hydrophobic interaction with the micelles- the stronger the interaction, the longer they take to migrate through the column with the micelle. Detection was by UV light at 254-nm wavelength.

Another CE method being used for PAH analysis is capillary electrochromatography (CEC). It is a combination of liquid chromatography (LC) and capillary electrophoresis. In CEC, the capillary is packed with a stationary phase similar to those used in LC. When an electric field is applied, the EOF moves the mobile phase through the packed column resulting in separation due to partition between the stationary and mobile phases. The separation is efficient and requires short analysis times. The CEC method has been used to separate the 16 priority PAHs, from a standard sample, on a fused-silica capillary packed with silica particles (DEH, 1999).

Currently, there are no instruments for measuring speciated PAH continuously in ambient air. Due to the low levels of these airborne compounds, it appears very unlikely that viable automated methods for speciated monitoring in ambient air will be developed in the near future. However, automated continuously recording devices have recently been developed (DEH, 1999) and utilized (Alberta Environment, 2004) for monitoring atmospheric concentrations of total PAHs. The photoelectric aerosol sensor (PAS) is an instrument used for real-time measurement of total particle-bound PAH concentrations in air (Agnesod et al., 1996). This instrument works on the principle of photoionization of the PAHs using an UV excimer lamp. The electrons emitted when the PAHs absorb the narrow band high intensity UV radiation are measured with an aerosol electrometer, the output signal of which is proportional to the total PAH concentration. The PAS sensitivity is in the ng/m^3 range. These instruments respond to a broad range of particle bound PAHs and produce a signal proportional to the combined concentrations of the suite of monitored PAHs. Although the instruments do not provide speciated PAH concentrations, they could be useful tools for identifying 'hot spots' for measuring PAH concentrations by conventional methods.

Passive samplers have been developed to measure concentrations of PAHs in air as an alternative to the standard active pump sampling techniques. These techniques have been applied only infrequently to ambient monitoring of PAHs, mainly because of the long sampling times required due to low PAH levels and limited analytical sensitivity (Peltonen and Kuljukka, 1995). The advantages of these samplers are that there are no moving parts to break down, regular flow calibration is unnecessary, and no bulky, expensive pumps are required. The sampler is exposed to ambient conditions for a set period of time (usually a much longer period than for active pump

sampling) and then analyzed by an appropriate analytical method (Brown and Wright, 1994; Levin and Lindahl, 1994). The PAH dosimeter utilizes a filter paper disc or a solid substrate treated with sorbent material such as a heavy-atom doped chemical reagent. This technique is convenient and sensitive but does not allow resolution of many components in the mixture. Passive biological sampling has also been investigated as an approach to long-term sampling of atmospheric PAHs (WHO, 1998). Correlation factors have been determined by comparing the PAH profiles in biological (i.e., plants) and air samples.

7.0 AMBIENT OBJECTIVES AND GUIDELINES

Current and/or recommended and proposed ambient guidelines of other jurisdictions in Canada, United States and elsewhere were reviewed for benzo[a]pyrene. In general, all jurisdictions have common uses for their guidelines. These uses may include:

- reviewing permit applications for sources that emit air pollutants to the atmosphere,
- investigating accidental releases or community complaints about adverse air quality for the purpose of determining follow-up or enforcement activity,
- determining whether to implement temporary emission control actions under persistent adverse air quality conditions of a short-term nature

7.1 Benzo[a]pyrene Air Quality Guidelines

Air quality guidelines for benzo[a]pyrene are summarized in Table 11. The principal approach by which guidelines are developed involves using carcinogenic risk assessment procedures. Pre-existing cancer risk assessments performed by others (e.g. US EPA Integrated Risk Information System summary data) are used to establish ambient air levels based on acceptable levels of lifetime cancer risk, such as one in 100,000 (10^{-5}) or one in 1,000,000 (10^{-6}). In addition some agencies have specific processes for estimating risks posed by exposure to mixtures of PAHs based on assumptions of additivity of individual risks posed by benzo[a]pyrene and other selected PAHs with four of more rings classified as carcinogens.

7.1.1 Canada

The Ontario Ministry of the Environment (MOE) adopted an Ambient Air Quality Criterion (AAQC) of 0.0011 $\mu g/m^3$ as a 24-hour guideline. Ontario MOE uses several maximum point of impingement (POI) guidelines: 0.0033 $\mu g/m^3$ from single sources for a 30-minute averaging time, 0.00022 $\mu g/m^3$ from single sources for an annual averaging time, and 0.0003 $\mu g/m^3$ from all sources also for an annual averaging time.

7.1.2 United States

No health-based air quality guidelines exist for benzo[a]pyrene from the US Agency for Toxic Substances and Disease Registry and US Environmental Protection Agency (US EPA). However, the US EPA's oral cancer slope factor of 7.3 $[mg/kg/day]^{-1}$ and an acceptable cancer risk level (e.g. 1 in 100,000 or 1 in 1,000,000) are used by a number of agencies to derive annual average guidelines (Arizona, Michigan, New Hampshire, and Washington). In addition, US EPA has a specific process for estimating risks posed by exposure to mixtures of PAHs based on assumptions of additivity of individual risks posed by benzo[a]pyrene and other selected PAHs with four of more rings classified as carcinogens.

Arizona, New Hampshire, and Washington have individual guidelines for other selected PAHs with four of more rings that are classified as carcinogens (commonly present as mixtures of PAHs in the atmosphere with benzo[a]pyrene). It is not stated how other PAHs with four of more rings classified as carcinogens (commonly present as mixtures of PAHs in the atmosphere with benzo[a]pyrene) are treated in Michigan.

A risk specific concentration (RsC) of 0.009 $\mu g/m^3$ corresponding to 1 in 100,000 risk is used to illustrate a benzo[a]pyrene guideline for the California Environmental Protection Agency (EPA). Cal EPA also has a specific process for estimating risks posed by exposure to mixtures of PAHs based on an inhalation unit risk of 1.1E-03 per ($\mu g/m^3$) for benzo[a]pyrene and assumptions of additivity of individual risks posed by other selected PAHs with four of more rings classified as carcinogens.

Louisiana, North Carolina, Texas, and Vermont have developed annual average guidelines for benzo[a]pyrene, however the derivation basis are unknown. The Louisiana guideline is to be applied to all PAH compounds with more than one fused benzene ring and which have a boiling point $\geq$100°C, excluding naphthalene and methylnaphthalenes. Texas has individual guidelines for other selected PAHs with four of more rings that are classified as carcinogens. It is not stated how other selected PAHs with four of more rings classified as carcinogens are treated in North Carolina and Vermont.

No guidelines exist for a number of states reviewed including Indiana, Massachusetts, New Jersey, Ohio, Oklahoma, Rhode Island, and Wisconsin.

7.1.3 International Agencies

The New Zealand Ministry of Environment and Ministry of Health recently proposed an air guideline for benzo[a]pyrene of 0.0003 $\mu g/m^3$ as an annual average. However, it is not stated how other selected PAHs with four of more rings classified as carcinogens are treated in New Zealand. The Netherlands National Institute of Public Health and the Environment (RIVM, 2001) do not have a guideline for benzo[a]pyrene.

The World Health Organization (WHO, 2000) recommended an ambient air guidance value of 0.0012 $\mu g/m^3$ for the general population using an inhalation unit risk factor of $8.7(10^{-5})$ per ng/m^3 and corresponding to an excess lifetime risk level of 1 in 100,000. The guideline is intended to provide background information and guidance to governments in making risk management decisions, particularly in setting standards. It is not stated how other selected PAHs with four of more rings classified as carcinogens are treated by the WHO.

Table 11 Summary of Air Quality Guidelines for Benzo[a]pyrene

Agency	Guideline Title	Guideline Value [µg/m³] Averaging Time:			
		1-hour	8-hour	24-hour	annual
Ontario MOE	Ambient Air Quality Criterion			0.0011	
	Maximum point of impingement Guideline -				
	From single sources:				0.00022
	From multiple sources:				0.0003
	Single sources (30-min. averaging time*):	0.0033*			
US ATSDR	No guideline exists.				
US EPA	No guideline exists.				
Arizona DHS	Arizona Ambient Air Quality Guideline				
	Risk specific concentration :[1]	0.67		0.18	0.00048
California EPA					0.009
Indiana DEM	No guideline exists.				
Louisiana DEQ	Ambient air standard:				0.06
Massachusetts DEP	No guideline exists.				
Michigan DEQ	Initial risk screening level:				0.0005
	Secondary risk screening level:				0.005
New Hampshire DES	24-hour ambient air limit :			0.005	
	Annual AAL:				0.005
New Jersey DEP	No guideline exists.				
North Carolina ENR	Acceptable ambient level:				0.000033
Ohio EPA	No guideline exists.				
Oklahoma DEQ	No guideline exists.				
Rhode Island DEM	No guideline exists.				
Texas CEQ	Effects screening level:	0.03			0.003
Vermont ANR	Hazardous ambient air standard				0.0003
Washington DOE	Acceptable source impact level:				0.00048
	ASIL for PAH mixtures:				0.00048
Wisconsin DNR	No guideline exists.				
New Zealand MOE	Air guideline (proposed):				0.0003
The Netherlands (RIVM)	No guideline exists.				
World Health Organization	Ambient air guidance value:				0.00012

[1] The RsC is not used for any specific purposes by the respective agency. It is shown here to illustrate an exposure concentration in air associated with an inhalation unit risk factor used by the agency and a 1 in 100,000 lifetime cancer risk (risk criteria used in Alberta).

8.0 RISK CHARACTERIZATION

Risk characterization of an ambient air contaminant involves the integration and evaluation of several types of key information. Where it is undertaken for a site-specific situation, such as for the screening assessment of proposed industrial emissions of benzo[a]pyrene, quantitative approaches can be used. For ambient air exposures, this may involve taking measurements or using a computer dispersion model to estimate ambient ground-level concentrations of benzo[a]pyrene under different averaging times and making comparisons to safe levels of exposure (air quality guidelines).

For the general case, however, use of site-specific risk characterization approaches is limited unless a number of assumptions are made. The emission characteristics for sources of benzo[a]pyrene release to air in Alberta vary for sources such as motor vehicle and industrial emissions. In addition, meteorological dispersion conditions can vary from site to site. This variability is difficult to represent as a general case for quantitative assessment and the resulting risk characterization would have substantial uncertainty. As a result, a qualitative risk characterization approach is used to evaluate issues associated with benzo[a]pyrene in ambient air. The types of key information evaluated can include, but not be limited to:

- relevant chemical forms in air
- pathways by which exposure may occur
- relevant potential effects (impacts)

These issues are discussed further below.

8.1 Relevant Chemical Forms

Benzo[a]pyrene is among a number of ≥3- to 4-ring PAHs that tend to be incorporated onto particulates during cooling and condensation in the atmosphere. Consequently, benzo[a]pyrene will mostly exist in the particle phase at normal ambient temperatures in the atmosphere. Particle sizes will be mostly ≤2.5 µm in aerodynamic diameter. These particles can be transported by wind and air currents until they return to the ground by wet or dry deposition.

8.2 Exposure Assessment

Dietary intake is the most important source of whole body exposure to PAHs for the general population (Thomson *et al.*, 1996; Butler *et al.*, 1993; Dennis *et al.*, 1991, 1983). Dennis *et al.* (1991, 1983) reported that the cereals group (i.e. bread, biscuits, cakes, etc.) and the oils/fats group contribute to up to 80% of total dietary intake of benzo[a]pyrene with fruit, sugars and vegetables providing much of the remainder.

Exposure to PAHs also occurs through the inhalation pathway, although in quantities much less than that received by dietary exposure (Kindzierski, 2000). Butler *et al.* (1993) reported that

average inhalation exposure to benzo[a]pyrene is 11 to 12 times less than food ingestion exposure for adults based on a dietary intake and personal air exposure monitoring study. The Canadian population spends a majority of time in indoor environments (~88% of time on average) with time spent inside the home being (~67% of time on average) being the most important based on time activity survey data (Leech *et al.*, 1996). This is likely a big factor why the most important setting where inhalation exposure occurs is inside the home. Other settings where inhalation exposure to benzo[a]pyrene and other PAHs with four of more rings classified as carcinogens are in the workplace and time spent in motor vehicles or near roadways.

With respect to the inhalation pathway, WBK (2004) summarized air concentration data for benzo[a]pyrene and other PAHs with four of more rings classified as carcinogens (commonly present as mixtures of PAHs in air with benzo[a]pyrene) from a number of studies. This included home indoor air, air outside homes, and urban (city) air (e.g. near roadways). The studies indicated that mean levels of these PAHs in urban air was more variable and tended to have higher concentrations than air inside and outside of homes. Greater variability in concentrations of benzo[a]pyrene in urban air is expected near sources such as roadways (motor vehicle emissions) and industrial sources (i.e. coke production, aluminum production, roofing, oil refining, and coal gasification operations) (ATSDR, 1995).

Occupational settings – such as coke production, aluminum production, roofing, oil refining, and coal gasification operations – are settings where exposures to benzo[a]pyrene and other PAHs with four of more rings classified as carcinogens may occur at amounts higher than home indoor air, air outside of homes, and in urban (city) air. These occupational exposures are in relation to emissions of coal tar pitch volatiles and mineral oil mists containing PAHs as mixtures (ATSDR, 1995).

8.3 Toxicity Assessment

Primary endpoints of toxicity associated with chronic, high benzo[a]pyrene exposures in animals are carcinogenicity, reproductive toxicity, and developmental toxicity. Other non-carcinogenic effects also reported at doses to animals know to be carcinogenic (growth inhibition, immunosuppression, liver damage, and irritation/hypersensitivities). Severity of the effects depends on dose, administration of dose (route and vehicle of administration), and animal species, age and genotype.

Epidemiological studies in coke-oven, coal-gas, and aluminum production workers have provided evidence of the role of inhaled PAHs in the causation of lung cancers. For example, the European Commission (2001) reported that increases in lung cancer cases were correlated closely with time spent by coke-oven workers on top of ovens where average benzo[a]pyrene concentrations of about 30 mg/m^3 have been detected. The amounts of benzo[a]pyrene associated with these types of effects are much greater than what occurs in indoor air, air outside of homes and in urban (city) air. Nevertheless, an important issue is the ability of inhaled benzo[a]pyrene and other PAHs with four of more rings to increase the risk of lung cancer in humans in non-occupational settings.

8.3.1 Mixtures of Carcinogenic PAHs

Inhalation exposure to single PAH compounds, e.g. benzo[a]pyrene alone, does not occur without other PAHs with four of more rings being present. Because several PAHs with four of more rings are treated as having the potential to cause cancer in addition to benzo[a]pyrene (WBK, 2004), an indicator for the carcinogenic fraction of these PAHs – present as mixtures in ambient air – is proposed to be benzo[a]pyrene. However, several methods for representing the potency of individual PAHs relative to benzo[a]pyrene have been published and are reproduced in Table 12 after WBK (2004).

Table 12 Relative Potency of Individual PAHs to Benzo[a]pyrene (Toxicity Equivalent Factor – TEF – Method)

Compound	CEPA[1]	MOEE[2]	US EPA[3]	Cal EPA[4]	RIVM[5]
naphthalene					
2-methylnaphthalene					
acenaphthylene					
acenaphthene					
fluorene					
anthracene		0.28			
phenanthrene		0.00064			0.01
acridine		0.0			
retene					
fluoranthene					0 to 0.06
pyrene		0.0			
1-nitropyrene					
benzo[a]fluorene				0.1	
benz[a]anthracene		0.014	0.1	0.1	0 to 0.04
7,12-dimethylbenz(a)anthracene					
benzo[c]phenanthrene		0.023			
chrysene		0.026	0.001	0.01	0.5 to 0.89
benzo[ghi]fluoranthene					
cyclopenta[cd]pyrene		0.012			
perylene					
benzo[b]fluoranthene	0.06	0.11	0.1	0.1	
benzo[j]fluoranthene	0.05	0.045		0.1	
benzo[k]fluoranthene	0.04	0.037	0.01	0.1	0.03 to 0.09
dibenz[a,h]anthracene		0.89	1.0		
benzo[a]pyrene	1.0	1.0	1.0	1.0	1.0
benzo[e]pyrene		0.0			
benzo[ghi]perylene		0.012			0.01 to 0.03
indeno[1,2,3-cd]pyrene	0.12	0.067	0.1	0.1	0 to 0.08
indeno[1,2,3-cd]fluoranthene					
dibenzo[a,h]pyrene		1.2		10	
dibenzo[a,i]pyrene				10	
dibenzo[a,l]pyrene				10	
coronene					
3-methylchloranthrene					

[1] Canadian Environmental Protection Act (EC, 1994).
[2] Ontario Ministry of Environment and Energy (OMEE, 1997).
[2] US Environmental Protection Agency (US EPA, 1993).
[3] California Environmental Protection Agency (Cal EPA, 1999b).
[5] The Netherlands (RIVM as cited in European Commission, 2001).

The Toxicity Equivalent Factor (TEF) method, indicated in Table 12 estimates individual PAH potencies relative to that of benzo[a]pyrene, in order to obtain a benzo[a]pyrene equivalent concentration (Albert *et al.*, 1983). The benzo[a]pyrene equivalent concentration can then be used to represent carcinogenicity of a mixture of PAHs in ambient air relative to the carcinogenicity of benzo[a]pyrene. Specifically, the concentration of each PAH classified as carcinogenic is multiplied by the appropriate TEF and then summed to provide an estimate of the benzo[a]pyrene equivalent concentration in ambient air.

Table 12 summarizes TEF values relative to benzo[a]pyrene for other PAHs classified as carcinogens by different organizations. As can be observed in Table 12, the choice of TEF method would result in different estimates of a benzo[a]pyrene equivalent concentration in ambient air based on PAHs present as mixtures.

To date the scientific literature has not attempted to rationalize and explain reasons for differences in TEF values, such as those observed in Table 12. However, several possible explanations are put forward:

- Scientific guidance clearly does not exist on the best or most defensible method in which determine cancer risks to hazardous chemicals, including mixtures of PAHs, when exposures are small.
- Principal dose-response data, which may include animal toxicological and/or human health epidemiological data, differs among the various approaches used for deriving TEFs.
- Criteria for weight of evidence of the available animal and/or human health effects information that is used for cancer risk assessment differs among the various methods used for deriving TEFs.
- Uncertainty related to causal association between disease and extrapolations to cancer risks posed by air pollution exposure are often interpreted differently among the various methods for deriving TEFs.

While the ultimate intent of each method is the same (i.e. derivation of TEFs for use in practice to provide protection of health and environment), differences exist in resulting TEF values derived.

Recommendation. As clear scientific guidance does not exist on the best or most defensible TEF derivation method, an interim method – in part after Crump (1995) – is recommended for setting an objective for PAH mixtures in ambient air for Alberta.

In animal-to-human extrapolation of non-cancer animal data, animal doses are frequently converted to equivalent human doses on a body weight basis (mg/kg/day), and additional factors

of 10 are applied (Crump, 1995). These factors are intended to allow the use of animal data to represent what may happen to humans, and to take into account that sensitive humans or subpopulations exist. In contrast, the current dose-response extrapolation methods for substances treated as potential carcinogens do not take into account differences between sensitive individuals or subpopulations and the healthy general population (Crump, 1995).

What is noted here is that regulatory toxicology applications make assumptions that dividing a threshold dose level for a substance from an animal study by factors of 10 results in an amount of the substance that is considered acceptable for exposure to people (Ziegler, 1993). These factors of 10 are used to account for the fact that some people might be more sensitive from exposure to a hazardous substance than animals and that different in sensitivities may exist among people exposed to the same amount of hazardous substance.

Crump (1995) has indicated that the "factors of 10" method for risk assessment is not based on a judgment that thresholds exist for most effects of both carcinogens and non-carcinogens. Rather, Crump (1995) indicated that it is based on a position that low-dose risks remain largely unknown for both carcinogenic and non-carcinogenic substances.

Therefore, an interim method using factors of 10 to represent the potency of individual PAHs relative to benzo[a]pyrene is recommended to address mixtures of carcinogenic PAHs in ambient air. Two examples are provided in Table 12 after US EPA (1993) and Cal EPA (1999). Either method should be suitable for the Alberta situation.

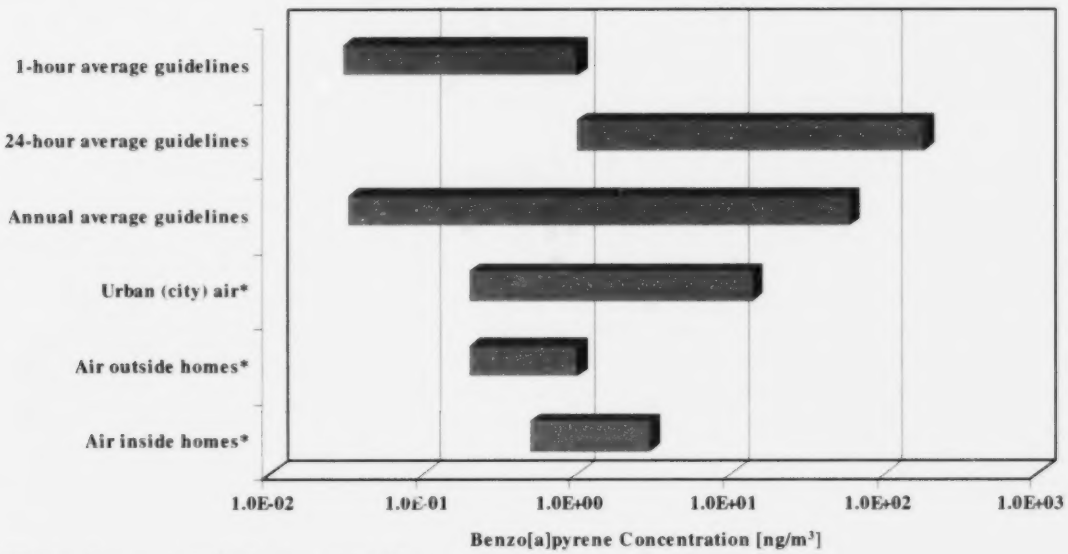
This recommendation does not presume that the most accurate, defensible scientific method to represent the potency of individual PAHs is being utilized (in fact the most accurate, defensible scientific method is yet unknown). Rather this recommendation is consistent with regulatory toxicology applications to aid in determining amounts of substances – in this case individual PAHs – that are considered acceptable for exposure to people. Further, this recommendation does not preclude use of an alternative scientific method for development of TEFs at a later date when warranted.

8.4 Characterization of Risk

An important concern to occupational health officials is the ability of inhaled benzo[a]pyrene and other PAHs with four of more rings to increase the risk of lung cancer in the workplace. These risks are of concern to coke production, aluminum production, roofing, oil refining, and coal gasification workers. The ability of benzo[a]pyrene and other PAHs to increase the risk of lung cancer in the general population in relation to low-level environmental exposures is much less certain. As a result regulatory agencies adopt air quality guidelines for benzo[a]pyrene and other PAHs in order to protect humans.

The range of air quality guidelines proposed by various agencies for protection of human receptors is shown in Figure 8-1 for different averaging time periods. The data presented in Figure 1 were summarized from Section 7.0.

Also shown in this figure are benzo[a]pyrene concentrations reported by WBK (2004) in indoor air and in air outside of homes and in urban (city) air. These concentrations do not take into account benzo[a]pyrene-equivalent concentrations contributed by other PAHs also present as a mixture in the air. Figure 8-1 illustrates that mean benzo[a]pyrene concentrations reported in indoor air, air outside of homes, and in urban (city) air are within the range of concentrations representing air quality guidelines for protection of human receptors.



*Range of mean concentrations as reported by WBK (2004)

Figure 1 Range of Air Quality Guidelines for Benzo[a]pyrene Proposed by Various Agencies for Protection of Human Receptors

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APPENDIX A

Table A-1 Emissions of Benzo[a]pyrene According to the 2001 NPRI Database(NPRI, 2004)

NPRI ID	Company	City	Province	Emissions of Benzo[a]pyrene (kg)				
				Air	Water	Land	Underground	Total
3903	Petro-Canada	Edmonton	AB	0.169	0	0	.301.543	301.712
3707	Imperial Oil	Edmonton	AB	1.515	0	14.910	0	16.425
2875	Weyerhaeuser Company Ltd.	Grande Prairie	AB	0.749	0	2.584	0	3.333
2274	Syncrude Canada Ltd.	Fort McMurray	AB	0.903	0	0	0	0.903
2960	Shell Canada Products	Fort Saskatchewan	AB	0.359	0	0	0	0.359
5357	Cancarb Limited	Medicine Hat	AB	0.2	0	0	0	0.2
403	Husky Oil Operations	Lloydminster	AB	0	0	0	0	0
2788	Alcan Primary Metal - British Columbia	Kitimat	BC	1300.3	1.380	0	0	1301.68
1797	Canadian Forest Products Ltd.	Prince George	BC	2.421	0	2.120	0	4.541
1383	Pope & Talbot Ltd.	Nanaimo	BC	1.074	1.132	1.518	0	3.724
1486	Pope & Talbot Ltd.	Mackenzie	BC	1.070	0	1.200	0	2.27
2377	Western Pulp Limited Partnership	Port Alice	BC	0.046	0	1.574	0	1.62
479	Cariboo Pulp and Paper Co.	Quesnel	BC	1.110	0	0.090	0	1.2
2924	Weyerhaeuser Company Limited	Kamloops	BC	1.140	0	0	0	1.14
723	Norske Skog Canada Limited	Powell River	BC	0.270	0.010	0	0	0.28
2776	Chevron Canada Limited	Burnaby	BC	0.132	0	0	0	0.132

Table A-1 Emissions of Benzo[a]pyrene According to the 2001 NPRI Database(NPRI, 2004) (continued)

NPRI ID	Company	City	Province	Emissions of Benzo[a]pyrene (kg)				
				Air	Water	Land	Underground	Total
1593	Norske Skog Canada Limited	Port Alberni	BC	0.002	0	0	0	0.002
7707	Greenwood Forest Products (1983) Ltd.		BC	0.001	0	0	0	0.001
4	Tembec Industries	Pine Falls	MB	1.261	0	0	0	1.261
2051	Tolko Manitoba Kraft Papers	The Pas	MB	0.571	0	0.021	0	0.592
1698	New Brunswick Power	Belledune	NB	124.679	0	0	0	124.679
1708	New Brunswick Power	New Castle Creek	NB	16.283	0	0	0	16.283
2604	Irving Pulp & Paper Limited / Irving Tissue Company		NB	1.670	0	8.060	0	9.73
1617	UPM-Kymmene Miramichi Inc.	Miramichi	NB	0.710	0	0	0	0.71
2181	St. Anne-Nackawic Company Ltd.	Nackawic	NB	0.511	0	0	0	0.511
4316	North Atlantic Refining Ltd.	Come by Chance	NL	0.024	0	0	0	0.024
3698	Imperial Oil	Dartmouth	NS	2.035	0	18 985	0	21.02
3992	Nova Scotia Power Inc.	New Waterford	NS	11.000	0	0	0	11
815	Kimberly-Clark Inc.	New Glasgow	NS	0.530	0	3.482	0	4.012
3713	Dofasco Inc.	Hamilton	ON	880	0	0	0	880
3855	Lake Erie Steel Company	Nanticoke	ON	814	0.048	0	0	814.048
2984	Stelco Inc.	Hamilton	ON	767	6.200	0	0	773.2
3704	Imperial Oil	Sarnia	ON	151.546	0	0	0	151.546

Table A-1 Emissions of Benzo[a]pyrene According to the 2001 NPRI Database(NPRI, 2004) (continued)

NPRI ID	Company	City	Province	Emissions of Benzo[a]pyrene (kg)				
				Air	Water	Land	Underground	Total
3701	Imperial Oil Limited	Nanticoke	ON	3.509	0	108.309	0	111.818
1070	Algoma Steel Inc	Sault Ste. Marie	ON	72.800	7.4	0	0	80.2
3071	Sunoco Inc.	Sarnia	ON	11.426	0	0	0	11.426
5798	Lafarge Canada Inc.	Woodstock	ON	10.700	0	0	0	10.7
3962	Shell Canada Products	Corunna	ON	4.693	0	2.877	0	7.57
1809	Ontario Power Generation Inc	Courtright	ON	0	0	6	0	6
3901	Petro-Canada	Oakville	ON	4.091	0	1.260	0	5.351
3185	Domtar Inc.	Espanola	ON	0.962	0	3.867	0	4.829
928	Weyerhaeuser Company Limited	Dryden	ON	1.148	0	1.102	0	2.25
2070	VFT Inc.	Hamilton	ON	2.100	0	0	0	2.1
1776	Nova Chemicals (Canada) Ltd.	Corunna	ON	0.620	0.011	0.975	0	1.606
1197	Domtar Papers	Cornwall	ON	1.531	0	0	0	1.531
3013	Norampac Inc.	Red Rock	ON	1.260	0	0	0	1.26
2607	Kimberly-Clark Corporation	Terrace Bay	ON	0.965	0	0	0	0.965
462	Marathon Pulp Inc.	Marathon	ON	0.456	0	0	0	0.456
3803	Ontario Clean Water Agency	Mississauga	ON	0.163	0	0	0	0.163

Table A-1 Emissions of Benzo[a]pyrene According to the 2001 NPRI Database(NPRI, 2004) (continued)

NPRI ID	Company	City	Province	Emissions of Benzo[a]pyrene (kg)				
				Air	Water	Land	Underground	Total
1236	Falconbridge Limited	Falconbridge	ON	0.140	0	0	0	0.14
1464	Imperial Oil	Sarnia	ON	0.116	0	0	0	0.116
5841	St. Marys Cement Inc.	Bowmanville	ON	0.110	0	0	0	0.11
5850	Lafarge Canada Inc.	Bath	ON	0.050	0	0	0	0.05
5871	St. Marys Cement Inc.	St Marys	ON	0.040	0	0	0	0.04
2182	St.Lawrence Cement	Mississauga	ON	0.005	0	0	0	0.005
5907	Northern Wood Preservers	Thunder Bay	ON	0.003	0	0	0	0.003
7072	Energy Plus 2000 Limited	Ajax	ON	0.001	0	0	0	0.001
2322	Crompton Co.	Elmira	ON	0	0	0	0	0
5623	Norbord Industries Inc.	Cochrane	ON	0	0	0	0	0
2844	Ontario Power Generation Inc.	Mississauga	ON	0	0	0	0	0
7081	Proboard Ltd.	Atikokan	ON	0	0	0	0	0
1054	Rhodia Canada Inc.	Lowbanks	ON	0	0	0	0	0
4268	Maritime Electric Company Limited	Charlottetown	PE	0.033	0	0	0	0.033
3406	Alcan Groupe Métal Primaire	Jonquière	QC	8789	0	0	0	8789
3057	Alcan Métal Primaire	Shawinigan	QC	3796	0	0	0	3796

Table A-1 Emissions of Benzo[a]pyrene According to the 2001 NPRI Database(NPRI, 2004) (continued)

NPRI ID	Company	City	Province	Emissions of Benzo[a]pyrene (kg)				
				Air	Water	Land	Underground	Total
2978	Alcan Bauxite	Jonquière	QC	0	0	3236	0	3236
4808	Alcan Métal Primaire	Melocheville	QC	1950.550	0	0	0	1950.55
2038	Alcoa Limitée	Baie-Comeau	QC	542.242	0.268	0	0	542.51
1071	Aluminerie de Bécancour inc.	Bécancour	QC	13.200	0	0	0	13.2
1195	Papier de Communication Domtar	Windsor	QC	1.605	0	5.26	0	6.865
3127	Produits Shell Canada	Montreal-est	QC	5.600	0	0.100	0	5.700
3897	Petro-Canada	Montreal	QC	4.352	0.002	0.003	0	4.357
5569	SGL Carbon Group	Lachute	QC	3.184	0	0	0	3.184
3928	Ultramar Ltée	St-Romuald	QC	0.829	0.888	0	0	1.717
1528	Papiers Fraser inc.	Thurso	QC	0.780	0	0	0	0.780
4782	Compagnie de Gestion Alcoa-Lauralco	Deschambault	QC	0.300	0	0	0	0.300
5513	Industries Manufacturières Mégantic inc	Lac-Mégantic	QC	0.022	0	0	0	0.022
2740	IPSCO Saskatchewan Inc.	Regina	SK	0.164	0	0	0	0.164
409	Husky Oil Operations	Lloydminster	SK	0	0	0	0	0

APPENDIX B

Agency: Ontario Ministry of the Environment (OME).
Air Quality Guideline: Ambient Air Quality Criterion (AAQC) = 0.0011 $\mu g/m^3$.
Averaging Time To Which Guideline Applies: 24-hour averaging time.
Basis for Development: Limiting effect based on health.
Date Guideline Developed: Unknown.
How Guideline is Used in Practice: Used by Ontario Ministry of Environment (OME) to represent human health or environmental effect-based values not expected to cause adverse effects based on continuous exposure.
Additional Comments: AAQC is not used by OME to permit stationary sources that emit benzo[a]pyrene to the atmosphere. A "point of impingement" standard is used to for permitting situations. It is not stated how other PAHs with four of more rings classified as carcinogens (commonly present as mixtures of PAHs in the atmosphere with benzo[a]pyrene) are treated.
Reference and Supporting Documentation: Ontario Ministry of the Environment. 1999. Summary of Point Of Impingement Standards, Point Of Impingement Guidelines, and Ambient Air Quality Criteria (AAQC). Standards Development Branch, Ontario Ministry of the Environment, Toronto, ON. November 1999. 12 pp.

Agency: Ontario Ministry of the Environment (OME).
Air Quality Guideline: Maximum point of impingement (POI) Guidelines: = 0.0033 µg/m^3 from single sources (30-minute averaging time) = 0.00022 µg/m^3 from single sources (annual averaging time) = 0.0003 µg/m^3 from all sources (annual averaging time)
Averaging Time To Which Guideline Applies: See above.
Basis for Development: Limiting effect based on health.
Date Guideline Developed: Unknown.
How Guideline is Used in Practice: Used by OME to review permit applications for stationary sources that emit benzo[a]pyrene to the atmosphere.
Additional Comments: It is not stated how other PAHs with four of more rings classified as carcinogens (commonly present as mixtures of PAHs in the atmosphere with benzo[a]pyrene) are treated.
Reference and Supporting Documentation: Ontario Ministry of the Environment. 1999. Summary of Point Of Impingement Standards, Point Of Impingement Guidelines, and Ambient Air Quality Criteria (AAQC). Standards Development Branch, Ontario Ministry of the Environment, Toronto, ON. November 1999. 12 pp.

Agency: US Agency for Toxic Substances and Disease Registry (ATSDR).
Air Quality Guideline: ATSDR does not have an ambient air guideline for this chemical.
Averaging Time To Which Guideline Applies: n/a
Basis for Development: n/a
Date Guideline Developed: n/a
How Guideline is Used in Practice: n/a
Additional Comments: US ATSDR develops minimum risk levels (MRLs) for hazardous chemicals. However, MRLs are based on noncancer health effects only and are not based on a consideration of cancer effects. Benzo[a]pyrene is treated as a human carcinogen.
Reference and Supporting Documentation: Agency for Toxic Substances and Disease Registry (ATSDR). 2004. Minimal Risk Levels (MRLs) for Hazardous Substances. ATSDR, Public Health Service, US Department of Health and Human Services. Atlanta, GA. Available at: http://www.atsdr.cdc.gov/mrls.html (accessed 17 February 2004).

Agency: US Environmental Protection Agency (US EPA).
Air Quality Guideline: US EPA does not have an ambient air guideline for this chemical.
Averaging Time To Which Guideline Applies: n/a
Basis for Development: n/a
Date Guideline Developed: n/a
How Guideline is Used in Practice: n/a
Additional Comments: US EPA has a specific process for estimating risks posed by exposure to mixtures of PAHs based on assumptions of additivity of individual risks posed by benzo[a]pyrene and other selected PAHs with four of more rings classified as carcinogens.
Reference and Supporting Documentation: US Environmental Protection Agency. 2004. Integrated Risk Information System. Available at: http://www.epa.gov/iris/ (accessed 17 February 2004). US Environmental Protection Agency. 1993. Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons. EPA/600/R-93/089. US EPA Office of Research and Development, Washington, D.C. July 1993.

Agency:
Arizona Department of Health Services (DHS).

Air Quality Guideline:
Arizona Ambient Air Quality Guidelines (AAAQGs):
Annual AAAQG = 0.00048 $\mu g/m^3$.
24-hour AAAQG = 0.18 $\mu g/m^3$.
1-hour AAAQG = 0.67 $\mu g/m^3$.

Averaging Time To Which Guideline Applies:
See above.

Basis for Development:
The annual AAAQG is derived by taking the US Environmental Protection Agency oral cancer slope factor of 7.3 $[mg/kg/day]^{-1}$ and an acceptable cancer risk of 1 in 1,000,000 (10^{-6}) using procedures described by Smith (1996).
The 24-hour AAAQG is derived by multiplying the annual AAAQG by 365.
The one-hour AAAQG is derived by multiplying the 24-hour AAAQG by 3.8. The multiplier of 3.8 represents the proportional difference in the lowest observed adverse effect level for 24-hour and 1-hour exposure to a common irritant (SO_2) in human subjects.

Date Guideline Developed:
Unknown.

How Guideline is Used in Practice:
AAAQGs are not intended to be used as standards. Rather, they are intended to provide health-based guidelines that may be useful in making environmental risk management decisions. AAAQGs consider human health risk from inhalation of contaminants in ambient air. They do not take into account odor thresholds or threats to wildlife.

Additional Comments:
AAAQGs are residential screening values that are protective of human health, including children. Chemical concentrations in air that exceed AAAQGs may not necessarily represent a health risk. Rather, when contaminant concentrations exceed these guidelines, further evaluation may be necessary to determine whether there is a true threat to human health.
Arizona DHS has individual guidelines for other selected PAHs with four of more rings that are classified as carcinogens (commonly present as mixtures of PAHs in the atmosphere with benzo[a]pyrene).

Reference and Supporting Documentation:
Arizona Department of Health Services (DHS). 1999. 1999 Update – Arizona Ambient Air Quality Guidelines (AAAQGs). Report prepared for Arizona Department of Environmental Quality, Air Programs Division. Arizona DHS, Office of Environmental Health, Phoenix, AZ. 11 May 1999. 20 pp.

Smith, R.L. 1996. Risk-Based Concentrations: prioritizing environmental problems using limited data. *Toxicol.* 106: 243-266.
US Environmental Protection Agency. 2004. Integrated Risk Information System. Available at: http://www.epa.gov/iris/ (accessed 17 February 2004).

Agency: California Environmental Protection Agency (Cal EPA).
Air Quality Guideline: Risk specific concentration (RsC) corresponding to 1 in 100,000 risk = 0.009 $\mu g/m^3$ (after rounding).
Averaging Time To Which Guideline Applies: Annual averaging time.
Basis for Development: The RsC corresponding to 1 in 100,000 risk (risk criteria used in Alberta) was derived as follows. Using respiratory tract tumor data from male hamsters, an inhalation unit risk of 1.1E-03 per ($\mu g/m^3$) was calculated using a linearized multistage procedure.
Date Guideline Developed: April 1999.
How Guideline is Used in Practice: The risk specific concentration (RsC) is not used for any specific purposes by Cal EPA and is shown here to illustrate an exposure concentration in air associated with an inhalation unit risk factor derived by Cal EPA and a 1 in 100,000 lifetime cancer risk.
Additional Comments: Cal EPA has a specific process for estimating risks posed by exposure to mixtures of PAHs based on the inhalation unit risk of 1.1E-03 per ($\mu g/m^3$) for benzo[a]pyrene and assumptions of additivity of individual risks posed by other selected PAHs with four of more rings classified as carcinogens.
Reference and Supporting Documentation: California Environmental Protection Agency (Cal EPA). 1999a. Determination of Acute Reference Exposure Levels for Airborne Toxicants. Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Section, Cal EPA. Oakland, CA. March 1999. California Office of Environmental Health Hazard Assessment (OEHHA)/Air Resources Board (ARB). 2004. Approved Chronic Reference Exposure Levels and Target Organs. Table 3 (last updated 4 December 2003). Available at: www.arb.ca.gov/toxics/healthval/chronic.pdf (accessed 17 February 2004). WBK & Associates Inc. (WBK). 2004. Approaches for Setting an Objective for Mixtures in Ambient Air Using Polycyclic Aromatic Hydrocarbons (PAHs) as an Example – Scoping of PAH Compounds. Report prepared for Alberta Environment, Edmonton, AB. February 2004. 29 pp.

Agency: Indiana Department of Environmental Management (IDEM).
Air Quality Guideline: IDEM does not have an ambient air guideline for this chemical.
Averaging Time To Which Guideline Applies: n/a
Basis for Development: n/a
Date Guideline Developed: n/a
How Guideline is Used in Practice: n/a
Additional Comments: n/a
Reference and Supporting Documentation: Indiana Department of Environmental Management (DEM). 2004. Office of Air Quality Programs. Indiana DEM, Office of Air Quality. Indianapolis, IN. Available at: http://www.in.gov/idem/air/programs/modeling/policy.html (accessed 17 February 2004).

Agency: Louisiana Department of Environmental Quality (DEQ).
Air Quality Guideline: Ambient air standard (AAS) for toxic air pollutants = 0.06 $\mu g/m^3$.
Averaging Time To Which Guideline Applies: Annual average.
Basis for Development: Not stated.
Date Guideline Developed: Not stated.
How Guideline is Used in Practice: AASs are used by Louisiana DEQ to review permit applications for stationary sources that emit benzo[a]pyrene and other PAHs treated as carcinogens to the atmosphere.
Additional Comments: Guideline is to be applied to all PAH compounds with more than one fused benzene ring and which have a boiling point greater than or equal to 100°C, excluding naphthalene and methylnaphthalenes.
Reference and Supporting Documentation: *Louisiana Administrative Code (LAC). Title 33 Environmental Quality, Part III Air, Chapter 51. Comprehensive Toxic Air Pollutant Emission Control Program. Louisiana Department of Environmental Quality. Baton Rouge, LA.*

Agency: Massachusetts Department of Environmental Protection (DEP).
Air Quality Guideline: Massachusetts DEP does not have an ambient air guideline for this chemical.
Averaging Time To Which Guideline Applies: n/a
Basis for Development: n/a
Date Guideline Developed: n/a
How Guideline is Used in Practice: n/a
Additional Comments: n/a
Reference and Supporting Documentation: Massachusetts Department of Environmental Protection (DEP). 1995. Revised air guidelines [updated list of 24-hour average Threshold Effects Exposure Limit (TEL) values and annual average Allowable Ambient Limit (AAL) values]. Massachusetts DEP, Boston, MA. 6 December 1995. Memorandum. Available at: http://www.state.ma.us/dep/ors/files/aallist.pdf (accessed 17 February 2004).

Agency: Michigan Department of Environmental Quality (DEQ).
Air Quality Guideline: Initial risk screening level (IRSL) = 0.0005 $\mu g/m^3$ [annual averaging time]. Secondary risk screening level (SRSL) = 0.005 $\mu g/m^3$ [annual averaging time].
Averaging Time To Which Guideline Applies: See above.
Basis for Development: The IRSL and SRSL are derived by taking the US Environmental Protection Agency oral cancer slope factor of 7.3 $[mg/kg/day]^{-1}$ and an acceptable cancer risk using procedures described by Smith (1996). The IRSL and SRSL are based on an acceptable cancer risk of 1 in 1,000,000 and 1 in 100,000 risk, respectively.
Date Guideline Developed: 1992.
How Guideline is Used in Practice: There are two basic requirements of Michigan air toxic rules. First, each source must apply the best available control technology for toxics (T-BACT). After the application of T-BACT, the emissions of the toxic air contaminant cannot result in a maximum ambient concentration that exceeds the applicable health based screening level for carcinogenic effects.
Additional Comments: There are two health-based screening levels for chemical treated as carcinogens by Michigan DEQ: the initial risk screening level (IRSL) – based on an increased cancer risk of one in one million, and the secondary risk screening level (SRSL) – based on as an increased cancer risk of 1 in 100,000. It is not stated how other PAHs with four of more rings classified as carcinogens (commonly present as mixtures of PAHs in the atmosphere with benzo[a]pyrene) are treated.
Reference and Supporting Documentation: *Michigan Administrative Code (MAC). Air Pollution Control Rules. Part 2 Air Use Approval, R 336.1201 - 336.1299. Air Quality Division, Department of Environmental Quality. Lansing, MI.* Michigan Department of Environmental Quality (DEQ). 2004. Air Toxics. Michigan DEQ, Air Quality Division, Lansing, MI. Available at: http://www.michigan.gov/deq/0,1607,7-135-3310_4105---,00.html (accessed 17 February 2004). Smith, R.L. 1996. Risk-Based Concentrations: prioritizing environmental problems using limited data. *Toxicol.* 106: 243-266. US Environmental Protection Agency. 2004. Integrated Risk Information System. Available at: http://www.epa.gov/iris/ (accessed 17 February 2004).

Agency: New Hampshire Department of Environmental Services (DES).
Air Quality Guideline: 24-hour ambient air limit (AAL) = 0.005 $\mu g/m^3$. Annual ambient air limit (AAL) = 0.005 $\mu g/m^3$.
Averaging Time To Which Guideline Applies: See above.
Basis for Development: Unknown. However the 24-hour and annual AAL are equivalent to taking the US Environmental Protection Agency oral cancer slope factor of 7.3 $[mg/kg/day]^{-1}$ and an acceptable cancer risk of 1 in 100,000 using procedures described by Smith (1996).
Date Guideline Developed: Unknown.
How Guideline is Used in Practice: AALs are used by New Hampshire DES to review permit applications for sources that emit benzo[a]pyrene to the atmosphere. Sources are regulated through a statewide air permitting system and include any new, modified or existing stationary source, area source or device.
Additional Comments: New Hampshire DES has individual guidelines for other selected PAHs with four of more rings that are classified as carcinogens (commonly present as mixtures of PAHs in the atmosphere with benzo[a]pyrene).
Reference and Supporting Documentation: *New Hampshire Administrative Rule. Chapter Env-A 1400. Regulated Toxic Air Pollutants. New Hampshire Department of Environmental Services. Concord, NH.* Smith, R.L. 1996. Risk-Based Concentrations: prioritizing environmental problems using limited data. *Toxicol.* 106: 243-266. US Environmental Protection Agency. 2004. Integrated Risk Information System. Available at: http://www.epa.gov/iris/ (accessed 17 February 2004).

Agency: New Jersey Department of Environmental Protection (DEP).
Air Quality Guideline: Applicants are required to carry out a risk assessment in conjunction with applying for an air pollution control pre-construction permit. In the case of benzo[a]pyrene, the US Environmental Protection Agency oral unit risk factor of 7.3 $[mg/kg/day]^{-1}$ and procedures described by Smith (1996) can be used to calculate a lifetime cancer risk for sources that emit benzo[a]pyrene to the atmosphere.
Averaging Time To Which Guideline Applies: Continuous (daily) exposure over a lifetime.
Basis for Development: Based on US EPA Integrated Risk Information System (IRIS) data.
Date Guideline Developed: December 1994.
How Guideline is Used in Practice: Used by New Jersey DEP to review permit applications for sources that emit benzo[a]pyrene to the atmosphere.
Additional Comments: It is not stated how other selected PAHs with four of more rings classified as carcinogens (commonly present as mixtures of PAHs in the atmosphere with benzo[a]pyrene) are treated.
Reference and Supporting Documentation: *New Jersey Administrative Code (NJAC). Title 7, Chapter 27, Subchapter 8. Permits and Certificates for Minor Facilities (and Major Facilities without an Operating Permit). New Jersey Department of Environmental Protection. Trenton, NJ.* New Jersey Department of Environmental Protection. 1994. Technical Manual 1003. Guidance on Preparing a Risk Assessment for Air Contaminant Emissions. Air Quality Permitting Program, Bureau of Air Quality Evaluation, New Jersey Department of Environmental Protection. Trenton, NJ. Revised December 1994. Smith, R.L. 1996. Risk-Based Concentrations: prioritizing environmental problems using limited data. *Toxicol.* 106: 243-266. US Environmental Protection Agency. 2004. Integrated Risk Information System. Available at: http://www.epa.gov/iris/ (accessed 17 February 2004).

Agency: North Carolina Department of Environment and Natural Resources (ENR).
Air Quality Guideline: Acceptable ambient level (AAL) = 0.000033 $\mu g/m^3$.
Averaging Time To Which Guideline Applies: Annual average.
Basis for Development: Unknown.
Date Guideline Developed: 1990.
How Guideline is Used in Practice: A facility emitting benzo[a]pyrene must limit its emissions so that the resulting modeled ambient levels at the property boundary remain below the health-based acceptable ambient level (AAL).
Additional Comments: It is not stated how other selected PAHs with four of more rings classified as carcinogens (commonly present as mixtures of PAHs in the atmosphere with benzo[a]pyrene) are treated.
Reference and Supporting Documentation: *North Carolina Administrative Code (NCAC). North Carolina Air Quality Rules 15A NCAC 2D.1100 – Air Pollution Control Requirements (Control of Toxic Air Pollutants). North Carolina Department of Environment and Natural Resources. Raleigh, NC.* *North Carolina Administrative Code (NCAC). North Carolina Air Quality Rules 15A NCAC 2Q.0700 – Air Quality Permit Procedures (Toxic Air Pollutant Procedures). North Carolina Department of Environment and Natural Resources. Raleigh, NC.*

Agency: Ohio Environmental Protection Agency (EPA).
Air Quality Guideline: Ohio EPA does not have an ambient air guideline for this chemical.
Averaging Time To Which Guideline Applies: n/a
Basis for Development: n/a
Date Guideline Developed: n/a
How Guideline is Used in Practice: n/a
Additional Comments: A number of PAHs with four of more rings, including benzo[a]pyrene are classified as B2 carcinogens by USEPA and applicants are required to perform a health impact/risk assessment study on these PAHs to determine maximum individual risks. If resultant maximum individual risks are greater than 10^{-5} per contaminant emitted, applicant is required to modify new source application and identify techniques to reduce maximum individual risks to less than 10^{-5} per contaminant.
Reference and Supporting Documentation: Ohio Environmental Protection Agency (EPA). 2004. Review of New Sources of Toxic Emissions. Air Toxics Unit, Division of Air Pollution Control, Ohio EPA. Columbus, OH. 11 pp (available at: http://www.epa.state.oh.us/dapc/atu/atu.html, accessed 17 February 2004). Ohio Environmental Protection Agency (Ohio EPA). 1994. Review of New Sources of Air Toxic Emissions. Proposed for Public Comment. Division of Air Pollution Control, Ohio EPA. Columbus, OH. January 1994. 31 pp.

Agency: Oklahoma Department of Environmental Quality (DEQ).
Air Quality Guideline: Oklahoma DEQ does not have an ambient air guideline for this chemical.
Averaging Time To Which Guideline Applies: n/a
Basis for Development: n/a
Date Guideline Developed: n/a
How Guideline is Used in Practice: n/a
Additional Comments: n/a
Reference and Supporting Documentation: *Oklahoma Administrative Code (OAC). Title 252. Chapter 100. Air Pollution Control. 100:252-41 - Control of Emission of Hazardous and Toxic Air Contaminants. Oklahoma Department of Environmental Quality. Oklahoma City, OK.* Oklahoma Department of Environmental Quality (DEQ). 2004. Air Toxics Partial Listing [maximum acceptable ambient concentrations (MAAC) for air toxics]. Oklahoma City, OK. Available at: http://www.deq.state.ok.us/AQDNew/toxics/listings/pollutant_query_1.html (accessed 17 February 2004).

Agency: Rhode Island Department of Environmental Management (DEM).
Air Quality Guideline: Rhode Island DEM does not have an ambient air guideline for this chemical.
Averaging Time To Which Guideline Applies: n/a
Basis for Development: n/a
Date Guideline Developed: n/a
How Guideline is Used in Practice: n/a
Additional Comments: n/a
Reference and Supporting Documentation: *Rhode Island Department of Environmental Management. 1992. Air Pollution Control Regulation No. 22. Division of Air and Hazardous Materials, Rhode Island Department of Environmental Management. Providence, RI. Amended 19 November 1992.*

Agency: Texas Commission on Environmental Quality (CEQ) – formerly Texas Natural Resource Conservation Commission (TRNCC).
Air Quality Guideline: Short-term effects screening level (ESL) = 0.03 $\mu g/m^3$. Long-term effects screening level (ESL) = 0.003 $\mu g/m^3$.
Averaging Time To Which Guideline Applies: 1-hour averaging time for short-term ESL. Annual averaging time for long-term ESL.
Basis for Development: Unknown.
Date Guideline Developed: Not stated.
How Guideline is Used in Practice: ESLs are used to evaluate the potential for effects to occur as a result of exposure to concentrations of constituents in air. ESLs are based on data concerning health effects, odor nuisance potential, effects with respect to vegetation, and corrosion effects. They are not ambient air standards. If predicted or measured airborne levels of a chemical do not exceed the screening level, adverse health or welfare effects would not be expected to result. If ambient levels of constituents in air exceed the screening levels, it does not necessarily indicate a problem, but rather, triggers a more in-depth review.
Additional Comments: Texas CEQ has individual guidelines for other selected PAHs with four of more rings that are classified as carcinogens (commonly present as mixtures of PAHs in the atmosphere with benzo[a]pyrene).
Reference and Supporting Documentation: Texas Natural Resource Conservation Commission (TNRCC). 2004. Toxicology & Risk Assessment (TARA) Section Effects Screening Levels. Available at: http://www.tnrcc.state.tx.us/permitting/tox/index.html (accessed 17 February 2004).

Agency: Vermont Agency of Natural Resources (ANR).
Air Quality Guideline: Hazardous ambient air standard (HAAS) = 0.0003 $\mu g/m^3$.
Averaging Time To Which Guideline Applies: Annual Average.
Basis for Development: The HAAS for known or suspected carcinogens (such as benzo[a]pyrene) is set at a level which represents an excess risk of one additional cancer case per one million exposed population (10^{-6}) assuming constant exposure at the HAAS concentration for a lifetime. However, it is unknown which unit risk factor was used to derive the HAAS.
Date Guideline Developed: Not stated.
How Guideline is Used in Practice: HAASs are used by Vermont ANR to review permit applications for stationary sources that emit benzo[a]pyrene to the atmosphere.
Additional Comments: It is not stated how other selected PAHs with four of more rings classified as carcinogens (commonly present as mixtures of PAHs in the atmosphere with benzo[a]pyrene) are treated.
Reference and Supporting Documentation: Vermont Air Pollution Control Regulations. 2001. State of Vermont Agency of Natural Resources. Air Pollution Control Division. Waterbury, VT. 29 November 2001. 187 pp.

Agency: Washington State Department of Ecology (DOE).
Air Quality Guideline: Acceptable source impact level (ASIL) = 0.00048 $\mu g/m^3$ for benzo[a]pyrene ASIL = 0.00048 $\mu g/m^3$ for mixtures of PAHs
Averaging Time To Which Guideline Applies: Annual average.
Basis for Development: ASIL for benzo[a]pyrene is derived by taking the US Environmental Protection Agency oral cancer slope factor of 7.3 $[mg/kg/day]^{-1}$ and an acceptable cancer risk of 1 in 1,000,000 using procedures described by Smith (1996). ASIL for mixture of other selected PAHs is same as for benzo[a]pyrene (specifically benzo[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, chrysene, dibenzo[a,h]anthracene, indeno(1,2,3-cd)pyrene, benzo[a]pyrene). These PAHs are to be considered equivalent in potency to benzo[a]pyrene.
Date Guideline Developed: Unknown.
How Guideline is Used in Practice: ASILs are used by Washington State DOE to review permit applications for sources that emit benzo[a]pyrene and other PAHs with four of more rings classified as carcinogens to the atmosphere.
Additional Comments: Applicant
Reference and Supporting Documentation: *Washington Administrative Code (WAC). Chapter 173-460 WAC. Controls For New Sources Of Toxic Air Pollutants. Washington State Department of Ecology. Olympia, WA.* Smith, R.L. 1996. Risk-Based Concentrations: prioritizing environmental problems using limited data. *Toxicol.* 106: 243-266. US Environmental Protection Agency. 2004. Integrated Risk Information System. Available at: http://www.epa.gov/iris/ (accessed 17 February 2004).

Agency: Wisconsin Department of Natural Resources (DNR).
Air Quality Guideline: Wisconsin DNR does not have an ambient air guideline for this chemical.
Averaging Time To Which Guideline Applies: n/a
Basis for Development: n/a
Date Guideline Developed: n/a
How Guideline is Used in Practice: n/a
Additional Comments: n/a
Reference and Supporting Documentation: *Wisconsin Administrative Code (WAC). Air Pollution Control Rules. Chapter NR 445. Control of Hazardous Pollutants. Wisconsin Department of Natural Resources. Madison WI.*

Agency: New Zealand Ministry for the Environment and New Zealand Ministry of Health.
Air Quality Guideline: Air guideline for protecting human health and well being = 0.0003 $\mu g/m^3$ (proposed).
Averaging Time To Which Guideline Applies: Annual average.
Basis for Development: Based on risk assessment and acceptable cancer risk of 2 to 3 in 100,000.
Date Guideline Developed: 2000.
How Guideline is Used in Practice: Air guidelines represent proposed guideline values for air-shed management.
Additional Comments: It is not stated how other selected PAHs with four of more rings classified as carcinogens (commonly present as mixtures of PAHs in the atmosphere with benzo[a]pyrene) are treated.
Reference and Supporting Documentation: New Zealand Ministry for the Environment and Ministry of Health (New Zealand). 2000. Proposals for Revised and New Ambient Air Quality Guidelines. Discussion Document. Air Quality Technical Report No 16. Prepared by the Ministry for the Environment and the Ministry of Health. December 2000. 79 pp.

Agency: The Netherlands National Institute of Public Health and the Environment (RIVM)
Air Quality Guideline: RIVM does not have air quality criteria for benzo[a]pyrene.
Averaging Time To Which Guideline Applies: n/a
Basis for Development: n/a
Date Guideline Developed: n/a
How Guideline is Used in Practice: n/a
Additional Comments: n/a
Reference and Supporting Documentation: The Netherlands National Institute of Public Health and the Environment (RIVM). 2001. Re-evaluation of human-toxicological maximum permissible risk levels. RIVN Report 711701 025. RIVN, Bilthoven, The Netherlands. March 2001. 297 pp.

Agency: World Health Organization (WHO)
Air Quality Guideline: Ambient air guidance value recommended for general population corresponding to an excess lifetime risk level of 1 in 100,000 = 0.00012 $\mu g/m^3$ (0.12 ng/m^3).
Averaging Time To Which Guideline Applies: Continuous (daily) exposure over a lifetime.
Basis for Development: The ambient air guidance value for benzo[a]pyrene based an increased cancer risk of 1 in 100,000 (10^{-5}) was derived by using an inhalation unit risk factor of $8.7(10^{-5})$ per ng/m^3.
Date Guideline Developed: 2000.
How Guideline is Used in Practice: The guideline is intended to provide background information and guidance to governments in making risk management decisions, particularly in setting standards.
Additional Comments: It is not stated how other selected PAHs with four of more rings classified as carcinogens (commonly present as mixtures of PAHs in the atmosphere with benzo[a]pyrene) are treated.
Reference and Supporting Documentation: World Health Organization (WHO). 2000. Air Quality Guidelines for Europe, 2nd Edition. WHO Regional Publications, European Series, No. 91. WHO Regional Office for Europe, Copenhagen. 273 pp.